

**AUTONOMIC AROUSAL AND
COGNITIVE PERFORMANCE
IN SEASONAL AFFECTIVE DISORDER**

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by

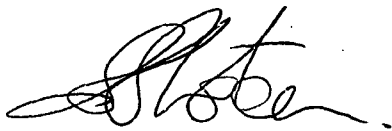
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For the degree of
Doctor of Philosophy

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May, 2003

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Margaret L. Austen

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30 May, 2003

Abstract

Seasonal affective disorder (SAD) has generally been seen to be at the extreme end of a continuum of seasonal variation to mood and associated behavioural symptoms in the general population. SAD is currently categorised in the DSM IV (American Psychiatric Association, 1994) as Seasonal Specifier, a type of recurrent disorder, which may be applied to (non-seasonal) Major Depressive Disorder (NSD). On the other hand, evidence is accumulating for SAD as a disorder distinct from NSD. Hence the thesis investigates the nature of SAD in relation to the seasonality of mood and behaviour in the general population. An epidemiological survey was conducted by administering the Seasonal Pattern Assessment Questionnaire (SPAQ) (Rosenthal, 1989) to Tasmanian Psychology 1 participants (N = 502) showing up to 9% and 24 % may have SAD at clinical (C-SAD) and subsyndromal (S-SAD) levels respectively. Statistical analyses on SPAQ variables further confirmed the short day aetiology of SAD and also implicated metabolic factors.

Two longitudinal studies were conducted, each using three groups (control, subsyndromal SAD, and clinical SAD) and six bimonthly testing sessions. Participants were screened using the SPAQ and selected according to Rosenthal's (1989) criteria. In each longitudinal study measures of depression, behavioural symptoms and emotion were also obtained at each testing session. Longitudinal Study 1 (n = 23 control, 21 S-SAD, and 18 C-SAD) investigated the proposition that the symptoms of SAD may represent an increased parasympathetic or decreased sympathetic arousal. Longitudinal Study 2 (n = 22 control, 21 S-SAD,

and 17 C-SAD) studied cognitive processing across the twelve-month period. Digit Span and Visual Memory Span subtests from the Wechsler Memory Scale measured memory processes and a Mental Rotation task as well as verbal and spatial versions of a Hemispheric Asymmetry Task determined cognitive efficiency, spatial processing, and any differential hemispheric specialisation effects that may be involved in SAD.

Distinctions between SAD and NSD were shown from the autumn/ winter specificity of the atypical vegetative behavioural symptoms accompanying decreases in mood and an underlying hypo-arousal showing similarities with hibernation. Impairments to cognitive processing include deficits specific to the spatial tasks that may have implications for differentiating between subsyndromal and clinical levels of SAD and also in understanding vulnerability to SAD. Implications for SAD theories are presented with findings indicative of dual underlying mechanisms consisting of a seasonal component, as well as a depression component in vulnerable SAD participants.

Circannual rhythms were documented and several variables were shown to vary with the seasons in control participants, thus extending current knowledge.

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Part 1

Introduction to seasonality

Chapter 1

Introduction to the investigation and overview of the thesis

Chapter 1: Introduction to the investigation and overview of the thesis

Seasonal variation to mood and behavioural symptoms is evident in a high percentage of the general population at latitudes of greater than 40° N or S.

Seasonal affective disorder (SAD) has recurrent autumn/ winter depressive episodes along with atypical vegetative symptoms, hypersomnia, increased appetite, weight gain, lack of energy, and social withdrawal. SAD is known to have a biological basis (e.g., Hill, 1992; Rosenthal & Wehr, 1992). The aetiology of SAD is thought to be the reduced daily photoperiod in the autumn and winter months that disrupts circadian rhythms and neurotransmitter production.

Light therapy, the accepted form of treatment in SAD, commenced after an anecdotal comment that additional bright light might have the effect of extending the hours of light to simulate a summer's day (Rosenthal, 2000). Light therapy is successful in the remission of symptoms, though a mechanism of action is unclear. A large body of experimental research has been conducted to determine the mechanism of action in an endeavour to determine a pathogenesis in SAD. While several theories have been put forward which add to understanding the pathophysiology in SAD, the picture is still unclear, and a pathogenesis unknown. It is also unclear as to which individuals might be susceptible to getting SAD.

Evidence suggests that a high percentage of the general population experience seasonal changes to mood and the atypical vegetative symptoms indicative of SAD (e.g., Kasper, Wehr, Bartko, Gaist, & Rosenthal, 1989). Therefore it appears that seasonal variation may represent a continuum of symptom

severity in the general population ranging from little or no seasonality, through mild to moderate changes, with SAD being an extreme of seasonal disturbances.

SAD is currently categorised in the Diagnostic and Statistic Manual of Mental Disorders (4th Edn.) (DSM IV) (American Psychiatric Association, 1994) as 'Seasonal Specifier' and may be applied to recurrent major depressive disorder. However some of the atypical symptoms of SAD (e.g., hypersomnia, increased eating, and weight gain) differ markedly from those typical in non-seasonal major depressive disorder (NSD) (e.g., insomnia, decreased eating, and weight loss). Further, a previous investigatory study by Austen and Wilson (2001) (copy of article in Appendix G) showed an *increase* in vagal tone during winter representing the sleep symptom. Hence, SAD may show similarities with the hibernation process thus implicating differing mechanisms from those in NSD. In contrast, vagal tone is typically *decreased* in NSD (Balogh, Fitzpatrick, Hendricks, & Paige, 1993; Rechlin, Weis, Spitzer, & Kaschka, 1994; Rechlin, Weis, Schneider, Zimmermann, & Kaschka, 1995). Thus the aim of this thesis is to determine the nature of SAD in relation to seasonality of atypical vegetative symptoms in the general population, seeking evidence for a distinction from NSD.

Chapter 2 introduces biological rhythms and gives an outline of their involvement in mood disorders. Chapter 3 outlines a brief history of seasonality in affective illness, commencing at 400 B.C. when Hippocrates believed that all disease was caused by the change in seasons, through the development of psychiatry in the eighteenth century, to current knowledge on seasonality and SAD.

The research approach to the thesis commences in chapter 4 with an epidemiological survey to determine the extent of seasonality, and degree of severity to seasonal changes in mood and associated behavioural symptoms. Tasmania, the southern most state of Australia, at latitudes of between 40° and 43.5° South is ideally situated to conduct the research. Various statistical procedures will be used to determine relationships among the variables.

Chapters 5 and 6 review the literature on SAD. Chapter 5 describes the clinical picture of SAD, also showing any psychological and physiological features that distinguish SAD from NSD. Light therapy is described and its efficacy examined. Additional treatments including drug therapy, psychological approaches, and lifestyle changes are also described. In order to determine a pathogenesis of SAD, a large body of experimental research has been conducted to determine the mechanism of action in light therapy. While the pathways are still unclear, several theories have been put forward which are reviewed in Chapter 6.

The experimental approach that was undertaken for the thesis consisted of two longitudinal studies: autonomic arousal (Chapter 7) and cognitive efficiency (Chapter 8).

Longitudinal study 1 investigates autonomic arousal in SAD at clinical and subsyndromal levels, and control participants, at bi-monthly intervals across a twelve-month period. The symptoms of SAD, for example, hypersomnia, increased appetite, weight gain, and lack of energy represent hypo-arousal that may indicate an increased parasympathetic or decreased sympathetic tone. Respiratory sinus

arrhythmia (RSA), a pure measure of parasympathetic nervous system activity, or vagal tone, has not previously been measured in SAD. Measures of sympathetic arousal, for example, heart rate, skin conductance level, and pulse wave transit time are also taken. Additional measures to support the study are blood pressure, sublingual temperature, skin temperature, body mass index, and percentage body fat. Mood, behavioural symptoms, and emotion are also measured at each bi-monthly testing session. The study suggests further similarities with hibernation, thus implicating differing underlying mechanisms from NSD.

Longitudinal study 2 examines cognitive efficiency in SAD at clinical and subsyndromal levels, and controls also across a twelve month period with six bi-monthly testing sessions. Memory problems and psychomotor deficits have been noted in SAD (e.g., Healy, 1987; Rosenthal, 1993). Two memory tasks, Digit Span and Visual Memory Span from the Wechsler Memory Scale - Revised (Wechsler, 1987) were administered to determine any disruption to memory in SAD at clinical and subsyndromal levels. Visuo-spatial deficits have been shown in SAD (Michalon, Eskes, & Mate-Kole, 1997; O'Brien, Sahakian, & Checkley, 1993) which may indicate pathophysiological problems in the retino-hypothalamic pathway. Furthermore, negative emotion is thought to originate in the right hemisphere. Two further tasks, Hemispheric Asymmetry and Mental Rotation were administered to determine any involvement of spatial tasks and/ or the right hemisphere. Mood, behavioural symptoms, and emotion were also measured at each bi-monthly testing session.

From the two longitudinal studies a body of knowledge on circannual rhythms will also be developed to add to current knowledge in the field. Cognitive efficiency is also determined in control group participants across the twelve-month period that may have implications for cognitive skills in the workplace, examination performance, and production efficiency during winter.

Chapter 9 will summarise the investigation, giving conclusions and future directions. Several conclusions are given. The importance of the metabolic symptoms in SAD shown from the epidemiological survey are consistent with suggestions of hypo-arousal from Longitudinal Study 1 implicating similarities with hibernation and as distinct from NSD. Secondly, deficits in memory and the Hemispheric Asymmetry tasks have implications for understanding any abnormality in the visual pathways as well as any hemispheric asymmetry specific to SAD. Thirdly, implications for current theories in SAD are discussed with support being given to a combination of mechanisms including metabolism and hypo-arousal. Overall, the findings suggest dual underlying mechanisms involving a seasonal factor shown from disturbances to sleep, energy, and weight, as well as a depression factor in those who are vulnerable.

Circannual rhythms for control participants are documented thus adding to current knowledge.

Chapter 2

An Introduction to Biological Rhythms, and

Seasonality in the General Population

Chapter 2: An Introduction to Biological Rhythms, and Seasonality in the General Population

"To everything there is a season and a time to every purpose under heaven ."

Ecclesiastes 3:1

Biological rhythms may underlie cyclic mood disorders with disruption of circadian and circannual rhythms (Hill, 1992). The aetiology of SAD is generally thought to be the shortened day length during winter. The onset on SAD occurs with the change in day length during the autumn.

Introduction

Biological rhythms are a regularly recurring quantitative change in a biological process. There are four biological rhythms or 'circa rhythms' that are, under normal conditions, synchronised with cycles in the environment. These four environmental cycles are the tides, day and night, the phases of the moon, and the seasons. The prefix 'circa' meaning 'about a' was prefixed to each of these cycles in the mid 20th century to give circa tidal, circadian, circa lunar, and circannual rhythms (Aschoff, 1981). Two essential features of biological rhythms that they are: (a) extrinsic in origin and depend on a regular change in the environment, for example, light or temperature, and (b) free running, that is, they persist even when environmental changes are absent (Aschoff; Kleitman, 1949). Two additional recurrent biological processes are known as *cycles* and *periodicity* that may also be referred to as rhythms. The terms rhythms, cycles, and periodicity have been used

interchangeably and applied to recurring fluctuations, oscillations, or variations. Cycles differ from rhythms in that they are intrinsic or endogenous in origin and vary among individuals. Heart rate, blood pressure, and respiratory sinus arrhythmia are examples of intrinsic cycles that may be influenced (but not caused) by either internal or external conditions. For example, heart rate may be influenced by internal physiology, and/ or external variations in the temperature. Periodicity, like the rhythm is extrinsic in origin, though differs in that it does not persist when external conditions are made uniform. Examples of periodicity are REM sleep or ovarian cycle (Kleitman).

Circadian rhythms characteristically free run and are synchronised with the light-dark cycle. However under some circumstances, may be influenced by environmental variables that disrupt their timing, creating a desynchronisation of physiological functions. These environmental variables are known as 'zeitgebers'. The most common zeitgebers for biological rhythms are light, temperature, variations in the weather, and social cues.

The biological pathway for circadian entrainment by light has been identified as the retino-hypothalamic tract, a monosynaptic pathway extending from the retina to the suprachiasmatic nucleus (SCN) of the anterior hypothalamus (Hill, 1992). The SCN is believed to be the endogenous pacemaker for circadian rhythms functioning primary by exerting a synchronising influence (Hill). The SCN innervates the pineal gland influencing production of melatonin, thus transducing the length of the daily photoperiod into biochemical language (Hill; Shafi & Shafi, 1990). Production of melatonin in the pineal gland is normally activated during darkness and suppressed by light during the day, and as such has been used as a

marker for depression, both in seasonal and non-seasonal depression. In SAD, melatonin is increased during winter due to an increase in duration of activation in the longer hours of darkness. In contrast, melatonin is low in NSD (Wetterberg, Beck-Friis, & Kjellman, 1990).

Because of the involvement of biological rhythms in seasonal affective disorder (SAD), this review on biological rhythms will be restricted to circadian and circannual rhythms. Both types of rhythms are a fundamental characteristic to most physiological, biochemical, and psychological function in humans. There is a direct relationship between circadian and circannual rhythms in that the amount of light, or daily photoperiod, varies more widely with the season in higher latitudes. Circannual rhythms are ongoing seasonal fluctuations that persist for more than one year or *cycle*. The formation of circannual rhythms is unclear, though the daily photoperiod is thought to be an important factor (Touitou & Haus, 1993).

A summary of biological rhythms indicates they are regularly recurring *cycles* that depend on changes in the environment and are capable of free running. Two types of biological rhythm, circadian or daily, and circannual or year long, rhythms may both have an involvement in seasonal affective disorder.

Biological rhythm involvement in mood disorders

Healy (1987) noted that disturbances of biological rhythms may underlie cyclic mood disorders, including seasonal and non-seasonal depressions. Circadian rhythms are known to show disruption with the differing photoperiod length at the change of seasons. In SAD, this internal rhythm could be phase delayed during the

winter months, or the result of an internal desynchronisation of circadian oscillators (Shafi & Shafi, 1990). For example, the tendency to sleep one hour longer would result in a one-hour phase delay. Melatonin production is influenced by the 24 hour light-dark cycle, with secretion stimulated during darkness and suppressed by light (Shafi & Shafi). During the longer hours of darkness in winter, an excess of melatonin is produced.

Any disruption in internal order can lead to further widespread disturbance to many physiological, biochemical, and psychological functions (Healy, 1987). For example, neurotransmitters in the retino-hypothalamic pathway are affected by the increased melatonin levels, further suppressing levels of serotonin. Further, Healy suggests that circadian rhythm disruption might lead to subtle disturbances in memory and learning, poor attention, loss of interest, loss of energy, loss of pleasure, and psychomotor retardation. Light therapy is specific to SAD. Simulating environmental light, exposure to bright light therapy has been used to phase advance or reset the internal desynchronisation, thus reversing the symptoms. Individuals who experience clear diurnal variations in mood have been shown to respond better to light treatment (Terman, Amira, Terman, & Ross, 1996) supporting the disruption of circadian rhythms in SAD and a synchronisation produced by light therapy. Drugs have also been shown to reset rhythms, having a phase adjusting or synchronising effect on the SCN (Healy; Hill, 1992).

A knowledge of circadian or circannual rhythm disruptions in any of the abovementioned variables, together with recent suggestions of a combined pathophysiology in SAD, is likely to be of assistance in further understanding SAD,

the underlying physiological mechanisms involved, and why some individuals are susceptible.

In summary, disruptions to biological rhythms may underlie cyclic mood disorders. In SAD, circadian rhythms are thought to be phase delayed during the winter months, thus creating desynchronisation and widespread disruption to physiological, biochemical, and psychological functions.

Most physiological and performance functions exhibit circadian rhythmicity and a large body of research documents these functions. For example, body temperature and heart rate (HR) peak in the evening and reach low values in the early morning. Vagal tone shows a diurnal rhythm increasing through the morning (Hayano et al., 1990). Food intake also affects vagal tone with a decrease in RSA after meals (Hayano et al.). On the other hand, sympathetic cardiac control shows little diurnal variation (Hayano et al.). Hormone levels in the blood show circadian rhythmicity. Noradrenaline is low during the day, being released during the night, and stimulating melatonin production. Peak concentrations of melatonin occur between two and four o'clock in the morning, while daytime concentrations are extremely low. Daytime concentrations of serotonin are greater than those measured at night (Hill, 1992; Shafi & Shafi, 1990). Performance efficiency measures show a rising level of performance throughout the morning, with a characteristic 'post-lunch' dip in performance.

At latitudes of greater than 40° N or S, the 24-hour light/ dark cycle varies considerably with the change in season. Hence, any circadian rhythmicity in human

functions may also vary with the seasons, giving a seasonal rhythm. Persistence of the seasonal rhythm for more than one cycle creates a circannual rhythm. Research documenting circannual rhythms has been limited due to (a) the intensive longitudinal nature of investigation required, and (b) the difficulty in controlling for methodological factors. Nevertheless a number of studies have investigated seasonality and circannual rhythms in the normal population as well as in SAD patients. For example, a review of empirical studies by Lacoste and Wirz-Justice (1989) indicates that several biological functions and behavioural patterns that have been considered important in the pathophysiology of SAD, also vary with seasons in the normal population.

Seasonal rhythms in the general population

Several variables that have an involvement in SAD, also show seasonality in the general population. It is possible that the general population adjust physiologically for winter onset, while those with SAD have an inappropriate response. Thus an understanding of seasonal rhythms in the general population may assist in isolating a pathogenesis and also in understanding why some people are more vulnerable to SAD than others.

Reports of a high level of winter mood, fatigue, and energy difficulties suggest a winter 'anergy' present in the general population which does not meet the criteria for SAD (e.g., Kasper et al., 1989; Terman, 1989). A tendency to sleep longer in winter, but not feel adequately rested has also been noted (Rosenthal et al., 1984). Increases in metabolic rates and body mass index have been reported for winter (Lacoste & Wirz Justice, 1989). Hypo-arousal (e.g., Skwerer et al., 1989;

Putilov & Danilenko, 1998) or a sluggish state of torpor has been noted in SAD during winter that may represent an energy conserving function. On the other hand, increases in energy expenditure are characteristic during the hypomania phase of SAD in the spring/ summer months.

Fluctuations in physiological variables are evident in control participants. A decrease in heart rate and increases in skin conductance level and diastolic blood pressure were shown for winter (Austen & Wilson, 2001). The P300 component of the event-related potential was shown to vary with the seasons, being smaller in autumn and winter than in spring which may have implications for cognitive performance (Deldin, Duncan, & Miller, 1994). Several physiological variables that have an involvement in thermo-regulation vary with the seasons and may be of assistance in understanding seasonal changes in mood and behaviour. Significantly cooler finger skin temperature (Gardner-Medwin, Macdonald, Taylor, Riley, & Powell, 2001) was reported for winter. The thyroid gland is required for an adjustment to temperature, with highest values for thyroxine and thyroid stimulating hormone for winter (Lacoste & Wirz-Justice, 1989). Blood glucose levels, involved in carbohydrate metabolism, are increased for winter (Lacoste & Wirz-Justice).

Seasonal disturbances to neurotransmitters and neuro-endocrine variables have been reported. Noradrenaline, dopamine, and serotonin are decreased during winter (Skwerer et al., 1989). Melatonin, secreted during darkness, is increased during winter with longer hours of darkness in a day. The hypothalamic pituitary adrenal (HPA) axis is known to have an involvement in depression with increases in adrenocortical activity (Lacoste & Wirz-Justice, 1989).

Mammals also experience biological rhythms. In most species of mammal, the hibernation process is an exogenous circannual rhythm that occurs spontaneously in response to a biological signal in the suprachiasmatic nucleus with the differing daily photoperiod length at the beginning of the cold season (Wehr et al., 2001). In other species however, the hibernation process is endogenous where the mammal responds to a shortage of food, and/ or the difficult environmental circumstances.

In summary, seasonal variation to many physiological, psychological, and biochemical measures is evident in the general population. Many of these measures have an involvement in seasonal affective disorder, and may be of assistance in understanding seasonal changes to mood and behaviour.

Conclusions

A biological rhythm is a regularly recurring quantitative change in a biological process. Two essential features of biological rhythms are that they are extrinsic in origin and depend on a regular change in the environment, and they free run, even when environmental changes are absent. Many of the circadian and circannual rhythms evident in the general population also have an involvement in SAD that may assist in understanding any underlying pathophysiological mechanism.

Chapter 3

Historical Overview of Seasonality in Affective Illness

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In early medical history, physicians emphasised the effect of the seasons on disease and, in particular, on melancholic depression and associated episodic mania. This chapter outlines the history of the effect of seasons on affective illness from approximately 400 B.C. when Hippocrates (460-370 B.C.) taught that all disease was caused by the change in seasons, through the development of psychiatry from the eighteenth century, to current knowledge on seasonality and seasonal affective disorder.

Early physicians and disease

Seasonal pattern in affective disorder has been recorded since 400 B.C. when Hippocrates suggested that most diseases were produced by the change in seasons, and in particular by the dramatic changes in temperature (Hippocrates, trans. 1959). Hippocrates taught the ancient Greek humoral theories of disease where melancholic depression was thought to result from a predominance of black bile occurring in autumn; while mania was caused by a predominance of yellow bile that occurred in summer. Aristotle (384-322 B.C.) also believed that seasonal changes in temperature acted on body humors (Aristotle, trans. 1953) causing disease and changes to mood. For example, he notes the effect of temperature on the humor black bile, quoting “black bile which is naturally cold produces torpor, or despondency, or fear . . . if the black bile became overheated, it produces cheerfulness, accompanied by song and frenzy” (p. 954).

Galen (129-200 A.D.), was an early physician who based his medical doctrine on Hippocrates' teaching. However, Galen disagreed with Hippocrates' belief that the change in seasons caused disease. Galen saw emotion to be the result of biological processes, with good health being an equilibrium of the humours (Galen, trans., 1973). He noted in seasonal depression. "... the symptoms arise together as a sympathetic affection. one type of sympathy persists only during the time of the formation (of the primary affection) and subsides together with the active cause" (p. 83). Galen may have been one of the first to report an association between lethargy in seasonal depression and hibernation (Galen, trans., 1976) when he stated "... cold weakens the mental activities. This is evident in animals which are forced by the frost to hibernate" (p. 81).

The ancient humoral theories were taught by Hippocrates over 2000 years ago. Early physicians consistently reported mania alternating with melancholic depressive episodes.

Recurrent seasonal pattern in depression

One of the earliest reported individual case studies with the pattern of winter melancholic depression and summer mania episodes was of an Englishwoman in the 17th century (Dewhurst, 1962). Her physician quoted "... there are twin symptoms, which are her constant companions, mania and melancholy, and they succeed each other in a double and alternate act" (p. 122). Her extreme seasonal mood swings included the mania phase of over-excitability, followed by depressive episodes where she was morose, lethargic, and expressed morbid delusions.

In the early 19th century, psychiatrists Pinel (1745 – 1826) and Esquirol (1772 – 1840) described a specific winter depression that may have been the earliest identification of an *actual syndrome* specific to a season, rather than merely the symptomatic expression of alternating mood change or illness in individual patients as had been known earlier (Wehr, 1989). Pinel (trans. 1962), known as the founder of modern psychiatry, noted that the seasonal influences on depression in one season were consistently reported as being associated with mania in the other season. The depressive episodes could be associated with either winter or summer, with mania in the opposite season. Seasonal depression was psychiatric in nature, with extreme mood swings and mania symptoms. Esquirol (trans., 1965) described the seasonal nature of affective illness in many patients, noting a milder form of seasonal affective illness. He quotes “It was necessary to live on terms of intimacy with her, in order to perceive the change” (p. 328).

In the early 20th century, Kraepelin (1856 – 1927) described recurrent winter depressions in individuals from the general population that were non-psychiatric (Kraepelin, 1921). He wrote

Repeatedly I saw in these cases moodiness set in in autumn and pass over in spring, when the sap shoots in the trees, to excitement, corresponding in a certain sense to the emotional changes which come over even healthy individuals at the changes of the seasons (p. 139).

Early physicians and psychiatrists noted the recurrent seasonal pattern of mania and melancholic depression. These depressive episodes could be in the summer or winter, with mania in the opposite season. A subsyndromal version of seasonal depression that affected healthy individuals from the general population was also reported.

Environmental influence on seasonal illness

Early physicians recognised that modification of the physical environment with temperature, warmth, and/ or light, could be used to treat affective episodes. Aristotle (trans., 1953) believed that heat elevated mood. Later Esquirol (trans., 1965) advised patients to travel to warmer climates for the winter. Others (e.g., Kraepelin, 1921; Pinel, trans., 1962) believed that warm baths gave excellent results. Galen, acknowledging that seasonal depression was the result of cooling of the brain, suggested that warming enabled its reactivation. The therapeutic effect of the sun was known in the 2nd century A.D., with exposure to the sun used as a therapy in lethargy (Wehr, 1989). Several references in the Bible also refer to the uplifting effect of light on mood, for example, “Truly the light is sweet, and a pleasant thing it is for the eyes to behold the sun” Ecclesiastes 11 : 7 (King James Version). The effect of light and darkness on mood have been written about by writers and poets (e.g., Henry Vaughan, Emily Dickinson, & T. S. Eliot) for centuries (Rosenthal, 1993). A biological basis for the reversal of symptoms has been observed. Emily Dickinson, in her poetry, recognised that her depression was due to the low angle of the sun in winter, affecting internal processes.

To summarise, it has been proposed that the influence of temperature, warmth, and light enabled the reversal of the symptoms in depressive episodes. The biological action of light on the eyes was noted centuries before discovery of the successful use of light therapy in the treatment of seasonal depression.

Decline in seasonal influence on disease.

Psychological and biological theories began to dominate psychiatric thinking from the mid-20th century. The ancient humoral theory, which had emphasised seasonal influences on disease, fell into disfavour and interest in seasonality decreased (Wehr, 1989). Psychiatrists began to treat affective recurrences as separate incidents, treating patients for their current symptomatic state, creating a shift away from a cyclical perception of time. Modern medical and psychological research has enabled a greater understanding of psychiatric illness. The mid-20th century development of psychotherapy together with the ongoing development of drugs (Garfield & Bergin, 1994) enabled the treatment of depression and mania states.

In summary, the greater understanding of psychiatric illness and advances in therapy over the past fifty years led to the decrease of interest in the cyclical nature of affective illness.

Interest in biological rhythms

A resurgence of interest in biological rhythms developed with the change of thinking in psychiatry that appears in early reports of seasonality, and the influence of the environment. Fluctuations of mood and behaviour are reported by a high percentage of the normal population (e.g., Kasper et al., 1989). Kasper reports energy activity in the normal population to be low in winter and high in summer that may be an attempt to conserve energy during winter. Seasonality has also been shown in many physiological and biochemical variables, as described in Chapter 2 (Lacoste & Wirz-Justice, 1989).

Seasonal affective disorder

In the early 1980s, Lewy and co-workers successfully treated a patient suffering from winter depression with light therapy (Wehr, 1989; Rosenthal, 2000). The patient was exposed to artificial light to extend the length of his day, thus simulating a summer day.

At the same time, Rosenthal and co-workers (Rosenthal et al., 1984) began to investigate several patients with winter depression at the National Institute of Mental Health (NIMH) in Maryland, USA. Rosenthal was the first to define SAD as a clinical entity. His diagnosis of SAD includes at least two consecutive years with autumn/ winter onset of depressive episodes and hypomania or remission of symptoms during spring/ summer. Symptoms accompanying the autumn/ winter depressive episodes include hypersomnia, overeating with carbohydrate craving, weight gain, and loss of energy. Diagnosis is exclusive of any psychosocial stressor that may cause the seasonal depressive episodes.

SAD, with its pattern of recurrent autumn/ winter depressive episodes, and spring/ summer remissions, along with the successful remission of symptoms with light therapy, appear to be similar to the cyclic episodes described by early psychiatrists, Pinel (trans., 1962) and Esquirol (trans., 1965). Furthermore, SAD sufferers acknowledge that the modification of their environment with temperature, warmth, and light, appear to be effective in relieving symptoms (Rosenthal, 1993). However, during remission from depression in SAD, sufferers are more likely to experience hypomania that is a less severe non-psychiatric mania (Rosenthal).

Since the early 1980s SAD has been documented extensively in locations of greater than 40° North or South latitude (e.g., Boyce & Parker, 1988; Helleckson, 1989; Dam, Jakobsen & Mellerup, 1998). Whilst the ætiology of SAD is unclear, SAD sufferers generally report their depressive episodes as relating to the short length of daylight during the autumn and winter months. SAD also involves autumn/ winter depressive episodes, hypersomnia, overeating with carbohydrate craving, weight gain, and loss of energy (Rosenthal et al., 1984). The recurrent depressive episodes appear similar to those described by early psychiatrists (Wehr, 1989). SAD sufferers acknowledge that modification of their environment assists in therapy.

Summary

Whilst the identification of SAD has occurred relatively recently, the effect of the seasons on disease, and seasonality in affective illness has played a major role for over 2000 years. Early physicians believed disease to be caused by the change of the seasons. Emphasis on the cyclical influence of disease declined in the mid-twentieth century due to changes in medical and psychological research. A resurgence of interest in seasonality in affective illness began in the 1980s when Lewy, Sack, Singer, White, and Hoban (1989) treated the first patient with light therapy, and Rosenthal et al.'s (1984) defining of the disorder known as "Seasonal Affective Disorder". Seasonality in mood and behaviour has been widely accepted over the past two decades, with extensive research having been conducted.

Chapter 4

An Epidemiological Survey of Seasonal Changes to Mood and Behavioural Symptoms in a Tasmanian University Sample

Chapter 4: An Epidemiological Survey of Seasonal Changes to Mood and Behavioural Symptoms in a Tasmanian University Sample

*"See! Winter comes, to rule the varied Year,
Sullen, and sad."*

from "Winter", James Thomson, 1726

Epidemiological findings from northern hemisphere locations of greater than 40° latitude suggest that mood and associated atypical vegetative symptoms indicative of SAD, also show seasonal changes to a lesser degree of severity in a high percentage of the population. The Seasonal Pattern Assessment Questionnaire (SPAQ) (Rosenthal, Kasper, Schultz, & Wehr, 1989), was developed as an instrument for evaluating retrospectively the degree and pattern of seasonal changes to mood and associated behavioural symptoms (see Appendix A). Scales for the six symptoms (mood, sleep, eating, weight, energy, and socialisation) determine the degree of seasonal change, with the sum of each symptom rating giving a Global Seasonality Score (GSS). The GSS has previously been validated as a measure of severity in SAD using a clinical diagnosis (Kasper et al., 1989; Terman, 1989). Participants also record the months that they feel best/ worst, gain/ lose weight, eat most/ least, sleep most/ least, and socialise least/ most, producing their seasonal profile. Additional subscales on the SPAQ include reactivity to climatic and atmospheric conditions, amount of weight fluctuation in a twelve-month period, and the average daily hours of sleep in each season. Finally, a global rating (GR) determines the participants' perception of seasonal change as a problem in their life. The GSS, the seasonal profile, and GR all contribute to the identification of SAD.

Studies of seasonality in the general population

Extensive studies from several locations were conducted in the 1980s using the SPAQ indicating that up to ninety percent of the general population report seasonal changes to mood and associated atypical vegetative behavioural symptoms to some degree. Studies administering the SPAQ to random samples in New York (latitude 40°) (Terman, 1989) and Maryland (latitude 39°) (Kasper et al., 1989) report greater percentages who experience variation to the atypical vegetative symptoms. For example, Terman noted 47% showed fatigue, 47% increased weight, 42% increased sleep, and decreased social activity; as compared to 31% mood. Kasper et al. found 90% of their sample reported seasonal change to mood and behaviour to some degree. Of these, 27% reported their seasonal change to be a problem. Further studies in London (Thompson, 1989), Switzerland (latitude 47°) (Wirz-Justice et al., 1989), and Alaska (latitude 64°) (Helleckson, 1989) show similar patterns confirming the winter prevalence of atypical vegetative symptoms of SAD in the general population.

In addition to the high incidence of seasonality in the general population, studies consistently report approximately eight to ten percent for C-SAD, and thirteen to eighteen percent for S-SAD that has also been known as 'winter blues', 'seasonal anergy syndrome', or 'winter complainers'. Hill (1992) conducted a random telephone survey and found fewer than 10% with SAD, and 13.5% with S-SAD. A study in Alaska (Helleckson, 1989) estimated 9% SAD and 18% S-SAD. Similar findings are reported estimating 9.5% SAD and 18.4% S-SAD in Finland (latitude 68-70°) (Saarijärvi, Lauerma, Helenius, & Saarilehto, 1999). Eagles et al.

(1999) estimated 2.9% SAD and 9.5% S-SAD in a study of psychiatric nurses in Scotland (latitude 57°). Similar percentages were found in Australian southern states Victoria and southern New South Wales (Boyce & Parker, 1988) even though these locations are less than latitude 40°. However, participants were predominantly women (87%) who responded to an article on seasonal mood changes in a national woman's magazine, limiting useful comparisons from the study and making it difficult to identify the specific latitudes involved. Most studies have used the SPAQ to estimate the prevalence of SAD. A lower rate of less than three percent with SAD was found in a Canadian study (latitude 44°) using a telephone interview methodology and restricting their sample to those with major depression according to DSM 111R (American Psychiatric Association, 1987), in addition to seasonal episodes (Levitt, Boyle, Joffe, & Bauman, 2000).

Overall, the studies suggest that in populations of greater than 40° north or south latitude, approximately eight to ten percent report SAD at a clinical level, and thirteen to eighteen percent report SAD at a subsyndromal level.

Prevalence rates according to latitude

The aetiology of SAD is thought to be day length with short days and a lesser amount of light during the autumn and winter months at locations of greater than latitude 40°. Because the days or the photoperiod, becomes shorter at higher latitudes, there is an assumption of a higher prevalence of SAD at higher geographic latitudes (Rosen & Rosenthal, 1991).

There are however exceptions to this generalisation of a higher prevalence of SAD at higher latitudes. Iceland, reports a lower prevalence of SAD than those obtained in the USA. Magnussen and Stefansson (1993) estimate the prevalence of SAD and S-SAD to be 3.8% and 7.5% respectively. Further research by Magnusson and Axelsson (1993) suggests that an increased tolerance of winter darkness at high latitude conditions over several generations may have contributed to these relatively low prevalence rates. In support of this explanation, high levels of S-SAD were found in short term research workers over winter in Antarctica.

Despite the exceptions that low levels of SAD have been reported at some high latitude locations, the weight of evidence indicates that prevalence of SAD increases in higher latitudes.

SAD: extreme of seasonal variation in the general population?

SAD has generally been seen as an extreme of seasonality in the general population suggesting a continuum of severity of the winter mood and energy difficulties, ranging from little or no seasonality, through mild to moderate seasonal symptoms, with SAD being an exaggeration of seasonal difficulties at the extreme end (e.g., Kasper et al., 1989; Terman, 1989). It is not clear why some individuals are vulnerable to greater seasonal disturbances than others. Because short daylight during the autumn/ winter months is a factor in SAD, one possibility is that some individuals may be more sensitive to the seasonal change of light.

Spring/ summer hypomania, or remission of symptoms?

A further controversial issue is whether SAD patients are predominantly bipolar or unipolar. Those sufferers who are predominantly bi-polar suffer recurrent depressive episodes during the winter months with hypomania in the summer or mild upswings of mood in the spring/ summer months (e.g., Helleckson, 1989; Rosenthal et al., 1989). These mild upswings in mood may be merely relief from the winter depressive episode, although similarities with bipolar disorder have been noted due to the recurrent cyclic nature of depressive episodes and mania/ hypomania (Thompson, 1989). In contrast, unipolar SAD sufferers experience the recurrent winter depressive episodes, with full remission during the summer (e.g., Lewy et al., 1989; Terman, 1989). Rosenthal et al. have suggested that latitudes and climates with more extreme swings in the strength of ambient light may predispose SAD patients to greater fluctuations in both directions.

In summary, some SAD sufferers experience hypomania during the spring/ summer months while others have a full remission of their symptoms.

In order to (a) to determine the extent of seasonal change to mood and associated atypical vegetative symptoms in a normal population, and (b) to determine the extent of SAD at clinical (C-SAD) and subsyndromal (S-SAD) levels, the SPAQ (Rosenthal et al., 1989) was administered to a Tasmanian population. Comparison will be made between the groups for each subscale on the SPAQ to validate participant classifications and to identify which of the GSS and GR measures best discriminates between the various groups. The study will also

determine any statistical structure from the SPAQ, for example evidence of bipolarity in symptoms and continuum of severity.

Method

Participants selection

Participants were Psychology 1 students ($n = 572$) from the University of Tasmania, Hobart. The Seasonal Pattern Assessment Questionnaire (SPAQ) (Appendix A); a questionnaire for screening seasonal change in mood and behaviour was completed by participants during their laboratory classes in the autumn of both 1995 and 1996. Analyses between the two years revealed no significant differences in seasonal variation to symptoms, global seasonality score (GSS), weight fluctuation (WtFluct), or global rating (GR), so the data for each year were combined. Participants were informed the study involved seasonal variation to mood and behaviour but specific seasons were not mentioned. Participants who had not lived in Tasmania for the previous three years ($n = 70$) were excluded leaving a total of 502 (females: $n = 361$ and males: $n = 141$, with ages ranging from 17 to 58 years).

The participants were placed in four groups. All those with a clear pattern of the symptoms in winter, a GSS greater than 13, and a GR of moderate or higher ($n = 49$, mean age = 21.31, SD = 6.80, range of 17 to 47), were categorised as clinical SAD (C-SAD). SPAQs with a clear pattern of winter symptoms, and a minimum GSS of 8 ($n = 133$, mean age = 22.47, SD = 8.61, range of 17 to 58), were

categorised as subsyndromal SAD (S-SAD). Controls included all those who experience little or no seasonality to mood and/or behavioural symptoms ($n = 124$, mean age = 21.68, $SD = 7.50$, range of 17 to 50). Because the SPAQ is neutral with respect to season, all those who recorded seasonality in months other than winter, and/or symptoms not indicative of SAD were classified as mixed seasonals (mixed) ($n = 196$, mean age = 20.43, $SD = 6.17$, range of 17 to 48) and included for example seasonal allergy sufferers, and those involved in seasonal sporting activity. A one-way ANOVA indicated there were no significant age differences between the groups. No cases of summer type seasonal affective disorder were identified in the Tasmanian sample.

Location. Tasmania, the smallest of Australia's states, is an island lying south of the southeast corner of the Australian mainland. The state has a total area of 68,331 km² and is separated from the Australian mainland by Bass Strait, a shallow sea with an average width of 240 km. Tasmania's population is 456,600. Approximately 40% of Tasmanians live in and around Hobart. Tasmania lies between latitudes 40° and 43.5° south and has a temperate maritime climate. Hobart is located at 42° 53' South latitude and 147° 20' East longitude with an elevation of 55.2 m. (Tourism Tasmania (On-line), 2001). Table 1 shows the variation in maximum and minimum temperature. The daily photoperiod for Hobart, Tasmania varies between 9 hours 01 minute and 15 hours 50 minutes for winter and summer solstices respectively. The mean photoperiod or hours of light in a twenty-four hour period, across a twelve-month period are also included in Table 1. Additional data in Table 1 are the daily hours of sunshine, and the mean number of clear, cloudy, and rainy days for each month.

Table 1

Mean Monthly Maximum and Minimum Temperatures (degree Celsius), Daily Photoperiod (hours and minutes), Daily Hours of Sunshine, and Mean Number of Clear, Cloudy, and Rain Days per Month, Calculated from Daily Recordings in Hobart, Tasmania

	January	February	March	April	May	June	July	August	September	October	November	December
	Mean											
Daily photoperiod (hours and minutes)	14.56	14.47	12.22	10.55	9.31	9.04	9.23	10.23	11.49	13.16	14.34	15.17
Daily maximum temperature (Deg C)	22.3	22.3	20.6	18.1	15.1	12.8	12.3	13.3	15.2	17.2	18.8	20.5
Daily minimum temperature (Deg C)	11.9	12.0	10.6	8.7	6.5	4.5	4.0	4.5	5.9	7.4	9.0	10.6
Daily sunshine (hrs)	8.1	7.8	6.6	5.7	4.6	4.2	4.7	5.5	6.1	7.1	7.3	7.7
No. of clear days	4.9	4.4	4.4	3.9	3.8	4.0	4.2	3.9	3.0	3.1	2.7	2.6
No. of cloudy days	13.5	11.5	13.5	14.0	15.7	14.3	14.7	14.5	14.3	14.6	15.6	15.9
No. of rain days	9.2	7.9	10.0	11.0	11.5	11.2	13.5	13.8	13.6	13.9	13.3	12.2

Note . Photoperiod was calculated from sunrise and sunset times for year 2000 (Australian Surveying and Land Information Group) (AUSLIG) (On-line) 2001). Climate averages were recorded at Hobart Airport over the past 43 years, and calculated on a daily basis (Bureau of Meteorology, (On-line) 2001). A clear day is recorded where the mean of the 9 am and 3 pm cloud cover is less than 2/8. A cloudy day is recorded where the mean of the 9 am and 3 pm cloud cover is greater than 6/8. A rain day is recorded when .2mm or more rain falls from in the 24 hour period from 9 am to 9 am

Australia, in the southern hemisphere has winter in June, July, and August, and summer in December, January, and February. Tasmania has daylight savings where clocks are put forward one hour from the first Sunday in October to the last Sunday in March (Tourism Tasmania (On-line), 2001).

Materials

Seasonal Pattern Assessment Questionnaire (SPAQ) (Rosenthal et al., 1989). The screening questionnaire, SPAQ is retrospective and self-rated, and obtains information according to the following categories:

1. Six-subscales (0-4) determine the participant's degree of seasonal change in sleep, social activity, mood, appetite, weight, and energy. (0 = no change, 1 = slight change, 2 = moderate, 3 = marked change, 4 = extremely marked change). This section is non-specific to a season, or any direction of change. The sum of the six scales gave a Global Seasonality Score (GSS) with a maximum score of 24.

2. The pattern of change for mood and behavioural symptoms determining any specific season and direction of change, and giving an overall seasonal profile.

Each participant is required to identify the month or months during which he or she feels best, feels worst, gains most weight, loses most weight, socialises most, socialises least, eats most, eats least, sleeps most, and sleeps least. A further column is available if the feeling/behaviour is not applicable in any month.

Seasonal profile data was entered into a spreadsheet as a 1 for each seasonal mood/behaviour the participant recorded the symptom for a particular month. The column was left blank where the feeling/behaviour was not applicable. The total score for each SAD symptom was subtracted from its opposite symptom (e.g., the total score

for 'feel worst' was subtracted from 'feel best') and converted to a percentage for each group.

3. Reactivity to ten different climatic and atmospheric conditions (cold, hot, humid, sunny, dry, long day, grey and cloudy, high pollen count, fog and smog, and short day) is rated. Participants give a rating (-3 to 3) that indicates the degree their mood/energy level changes with different weather conditions. (0 = no effect, with negative scores being either slightly, moderately, or very low spirits/ slowed down and positive scores either slightly, moderately, or markedly improving mood or energy level).
4. The participant records their amount of weight fluctuation in a twelve month period on a scale from 1 to 6 (1 = 0-2 kg, 2 = 2-3 kg, 3 = 4-5 kg, 4 = 6-7 kg, 5 = 8-10 kg, 6 = over 10 kg).
5. The participant estimates the average number of hours of sleep (to the nearest half hour) in a 24-hour period for spring, summer, autumn, and winter.
6. Each participant records a global rating (GR) (0-5) indicating their perception of seasonal change as a problem in their life (0 = not a problem, 1 = mild, 2 = moderate, 3 = marked, 4 = severe, 5 = disabling).

The SPAQ has previously been shown to be a valid instrument in measuring seasonality and has been widely used in epidemiological studies in the general population (e.g., Kasper et al., 1989; Terman et al., 1989) as well as in clinical samples (Rosenthal et al., 1984; Thompson, 1989). Test-retest reliability for the GSS has been shown to be high in several studies (e.g., Hardin et al., 1991; Terman et al.; Thompson). A factor analysis conducted by Magnusson, Friis, and

Opjordsmaan (1997) shows the SPAQ to have a high internal consistency with a Cronbach alpha of 0.82.

Data analyses

Seasonal Profiles. The percentage of each group experiencing a mood/ or behaviour for each month was recorded.

Analyses on SPAQ variables. Univariate between groups ANOVAs were conducted separately between the four groups (C-SAD, S-SAD, non-SAD seasonal, and non-seasonal control) with global seasonality score (GSS), global rating (GR), seasonal weight fluctuations (WtFluct), and age, as dependent variables.

Multivariate analyses using Pillai's Trace criterion were conducted on all remaining SPAQ variables. A one-way between groups MANOVA was conducted on six dependent variables. These were the degree of change experienced across a twelve-month period to six mood and behavioural symptoms: appetite, energy level, mood, sleep, socialisation, and weight. A further one-way between groups MANOVA was conducted on ten dependent variables. These were the degree to which participants' mood/ energy levels changed with ten differing weather patterns: cold, hot, humid, sunny, dry, grey and cloudy, long daylight, high pollen count, fog and smog, and short daylight. Follow up univariate analyses were conducted on each dependent variable for significant one-way MANOVAs. A 4 x 4 (Group x Season) repeated measures MANOVA was conducted on the mean number of hours of sleep recorded for each season with Group (C-SAD, S-SAD, non-SAD seasonal, and non-seasonal control) as a between groups factor and Season (spring, summer, autumn, and

winter) as a within subjects factor. LSD post hoc analyses were conducted to locate significant effects where appropriate according to Tabachnik and Fidell (1989). The significance level was set at $\alpha = .05$.

Principal components analysis. A principal components analysis with varimax rotation was performed to (a) reduce the complexity of the large number of variables, and (b) to determine any underlying structure in the SPAQ for the Tasmanian population. All data from the SPAQ were included in the principal components analysis giving a total of 35 variables. These were the six symptom subscales (appetite, energy, mood, sleep, socialisation, and weight), GSS, 12 monthly composite symptom scores, reactivity to ten climatic and atmospheric conditions (cold, hot, humid, sunny, dry, grey/cloudy, long daylight, high pollen count, fog/smog, and short daylight), degree of weight fluctuation, mean hours of sleep for each season, and GR. Visual inspection of the scree plot was used to determine the number of factors following the principal components analysis according to Tabachnik & Fidell's (1989) criteria.

Discriminant analysis. A hierarchical discriminant function analysis was performed on C-SAD, S-SAD, and controls to assess (a) the best prediction of group membership from SPAQ variables, (b) the degree of association between group membership and the predictors by investigating the number of reliable discriminating functions between the groups. The mixed seasonal group was excluded from the analysis due to its mixed seasonal pattern, and non-SAD like symptoms.

Multiple regression analyses. Multiple regression analyses using the stepwise method were performed to investigate relationships between SPAQ variables, in particular between the six symptoms, ten climatic conditions, GSS, and GR. The relative importance of each symptom as well as climatic conditions to SAD and aetiology will be determined.

Results

Seasonal Profiles

The percentage of each group recording a symptom for a particular month is shown in Figure 1. A tendency for all groups to feel worst, sleep longer, socialize least, have greater appetites, and gain weight during the winter months is evident from each symptom in the figure. Figure 1 also shows a tendency for the groups to feel best, sleep least, socialise most, eat least, and lose weight during the spring and summer months. From Figure 1, higher percentages of both SAD groups show these mood and behavioural symptoms for autumn/ winter, and greater levels of the opposite of each symptom, rather than a remission, during the spring and summer months.

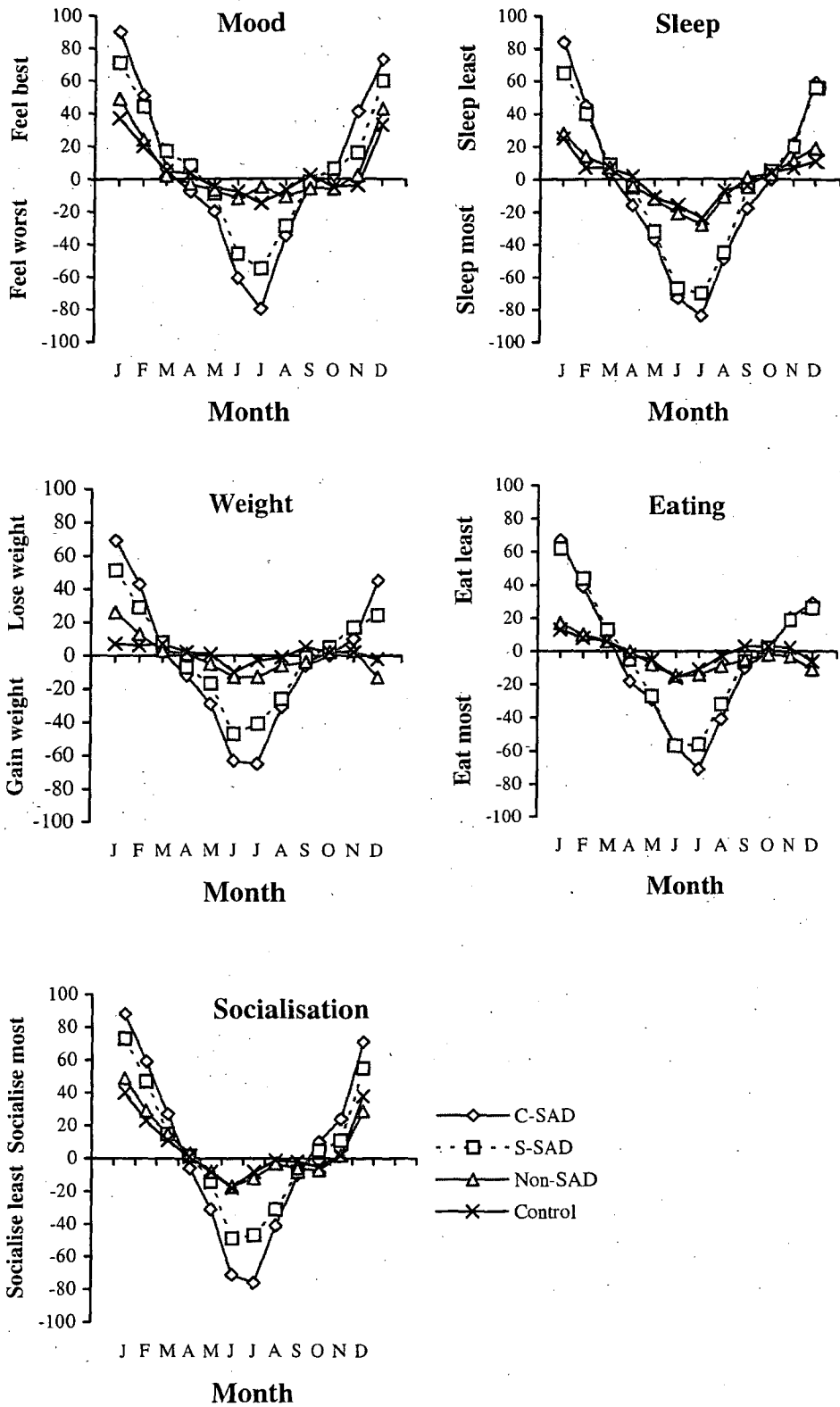


Figure 1. Seasonal profiles for each group experiencing a symptom for a particular month. SAD symptoms, feel worst, sleep most, gain weight, eat most, and socialise least, are represented below the x-axis

Univariate analyses

Means and standard deviations for the SPAQ variables are presented in Table 2 for the four groups, C-SAD, S-SAD, mixed seasonals, and controls.

One-way between groups ANOVAs were performed separately between the four groups (C-SAD, S-SAD, mixed seasonals, and controls) with global seasonality score (GSS), global rating (GR), weight fluctuation (WtFluct), and age, as dependent variables. Main effects were revealed for both GSS, $F(3, 498) = 252.04, p < .0001$, and GR, $F(3, 498) = 54.27, p < .0001$, indicating significant differences in ratings between the groups. Post hoc *t*-tests indicated the C-SAD group to have significantly higher GSSs and GRs than S-SADs, mixed seasonals and controls, at the .001 level of significance. S-SAD and mixed seasonal groups did not differ from each other on either rating.

A One-way ANOVA was performed between groups for the degree of WtFluct during a twelve month period revealing a main effect for Group, $F(3, 496) = 15.30, p < .0001$. Post hoc analyses revealed significant differences in ratings between all pairwise comparisons except S-SAD and mixed seasonals. The C-SAD group reports a significantly greater degree of WtFluct at the $p = .001$ significance level than S-SADs, mixed seasonals and controls. Table 2 shows the mean WtFluct for each group.

Table 2

Means, and Standard Deviations for Self-rated Scores on the Screening Questionnaire (Seasonal Pattern Assessment Questionnaire) for Control, Mixed Seasonal, Sub-syndromal SAD (S-SAD), and Clinical SAD (C-SAD) groups

	Source	Controls		Mixed seasonals		S-SAD		C-SAD	
		M	SD	M	SD	M	SD	M	SD
Degree of seasonal change to:	Appetite	0.68	0.69	1.80	1.12	2.01	0.89	2.72	0.82
	Energy	0.90	0.75	2.21	0.90	2.26	0.85	3.13	0.54
	Mood	0.82	0.82	1.74	1.02	1.88	0.90	2.82	0.78
	Sleep	0.94	0.76	1.87	0.87	1.91	0.88	2.74	0.77
	Social	1.12	0.89	2.05	0.97	2.10	0.97	2.86	0.88
	Weight	0.41	0.65	1.30	1.05	1.45	0.94	2.37	0.89
Global Seasonality Score		4.85	2.09	10.95	3.30	11.57	2.71	16.64	2.25
Global Rating		1.11	0.60	1.60	0.89	1.66	0.77	2.78	0.71
Weight Fluctuation		1.56	0.91	2.10	1.02	2.06	0.90	2.60	1.11
Sleep Length	Autumn	7.93	1.13	8.21	1.09	8.24	1.24	8.47	1.09
	Winter	8.27	1.21	8.75	1.26	9.13	1.39	9.90	1.24
	Spring	7.69	1.10	7.88	1.10	7.79	0.91	7.83	0.90
	Summer	7.60	1.24	7.48	1.41	7.29	1.07	7.04	1.06
Age		21.68	7.50	20.43	6.17	22.47	8.61	21.31	6.80

Note. The degree of change in a twelve month period irrespective of season for each symptom ranged from 0 (no change) to 4 (extremely marked change) gave the Global Seasonality Score was the sum of the six symptoms. Global rating ranged from 0 (mild) to 4 (severe) and was the participant's overall perception of seasonality disturbance felt. Weight fluctuation (1-6; 1 = 0-2 kg, 6 = over 10 kg) was the degree to which participant's weight fluctuates across a year. Sleep length was the approximate hours slept per day in each season.

N =

Summary of the univariate variables. The groups were differentiated with higher scores for GSS, GR, and WtFluct, for both the SAD groups than for controls. C-SADs had higher scores than S-SADs.

Degree of change across season to feelings/behaviours

A one-way between groups MANOVA performed on six dependent variables: degree of change across season to appetite, energy, mood, sleep, social activity, and weight was significant, Pillai's Trace = 0.63, $F(18, 1461) = 21.67$, $p < .0001$. Subsequent univariate F tests on each dependent variable showed all symptoms to contribute to significant differences between the groups. Univariate F values are: appetite, $F(3, 490) = 72.37$, $p < .001$, energy, $F(3, 490) = 114.12$, $p < .001$, mood, $F(3, 490) = 62.21$, $p < .001$, sleep, $F(3, 490) = 64.60$, $p < .001$, socialisation, $F(3, 490) = 47.75$, $p < .001$, and weight, $F(3, 490) = 59.58$, $p < .001$.

Post hoc pairwise comparisons for all groups conducted for each symptom separately showed higher significant differences at the $p < .001$ level, except between S-SAD and mixed seasonals. From Figure 2, C-SADs rate each symptom significantly higher than S-SADs, mixed seasonals, and controls. S-SADs and mixed seasonals also rate each symptom significantly higher than controls.

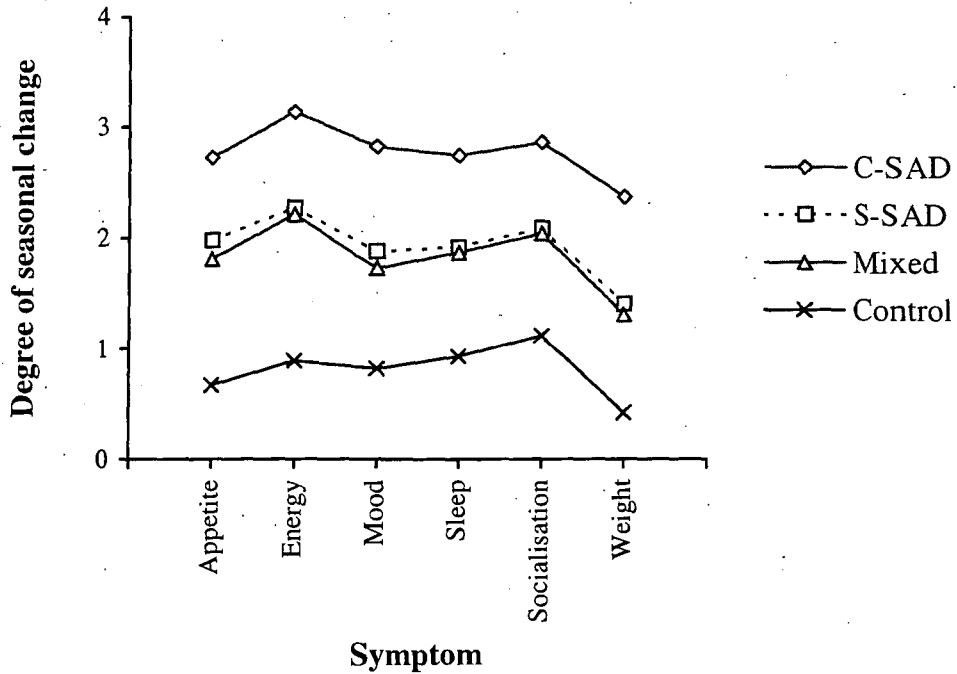


Figure 2. Degree of change in a twelve month period irrespective of season for clinical SAD (C-SAD), subsyndromal SAD (S-SAD), mixed seasonal, and control groups for six symptoms on the Seasonal Pattern Assessment Questionnaire

Summary of results from degree of seasonal change. The SPAQ differentiated between the groups for degree of seasonal change in each of the six symptoms. The two SAD groups rated all symptoms higher than controls with C-SADs rating symptoms higher than S-SADs. Mixed seasonals did not differ from S-SADs.

Reactivity to atmospheric and climatic conditions

Means and standard deviations for the degree to which participants' mood and energy levels change with different weather conditions are presented in Table 3.

Table 3

Means and Standard Deviations for the Degree of Change to Mood and Energy Levels in Response to Ten Weather Conditions (Cold, Hot, Humid, Sunny, Dry, Grey and Cloudy, Long Daylight, High Pollen Count, Fog and Smog, and Short Daylight) for Clinical SAD (C-SAD), Subsyndromal SAD (S-SAD), Mixed Seasonal (Mixed), and Control Groups

Group	Cold	Hot	Humid	Sunny	Dry	Grey/Cloudy	Long day	High pollen	Fog/ Smog	Short day
Means (SD)										
C-SAD	-1.93 (1.11)	1.05 (1.92)	-0.36 (1.45)	2.48 (1.17)	0.95 (1.22)	-1.45 (1.02)	2.18 (0.90)	-0.77 (1.22)	-1.52 (1.21)	-1.68 (0.83)
S-SAD	-1.41 (1.07)	0.88 (1.58)	-0.62 (1.37)	2.13 (1.02)	0.50 (1.15)	-1.18 (0.94)	1.83 (1.10)	-0.67 (1.04)	-1.36 (0.98)	-1.10 (0.95)
Non-SAD	-0.56 (1.57)	0.17 (1.70)	-0.81 (1.47)	1.75 (1.33)	0.25 (1.20)	-0.69 (1.25)	1.56 (1.23)	-0.69 (1.29)	-1.07 (1.07)	-0.95 (1.19)
Control	-0.52 (1.29)	0.18 (1.56)	-0.76 (1.27)	1.26 (1.26)	0.24 (1.03)	-0.45 (1.00)	1.13 (1.22)	-0.30 (0.89)	-0.78 (1.09)	-0.57 (0.87)

Note . Participants marked the degree to which each weather condition affected their mood/ energy level. Ratings were made on a 7-point scale where -3 = makes you feel in very low spirits or markedly slowed down, 0 = no effect, 3 = markedly improves your mood or energy level. n = 44 C-SAD, 115 S-SAD, 175 mixed seasonals and 115 control participants.

A one-way between groups MANOVA performed on ten dependent variables: cold, hot, humid, sunny, dry, grey and cloudy, long day, high pollen count, fog and smog, and short day, was significant, Pillai's Trace = 0.26, $F(30, 1314) = 4.15$, $p < .001$. Follow-up univariate F tests on each dependent variable showed all weather conditions, except for humid, produce significant differences between the groups. Univariate F values were: cold, $F(3, 445) = 20.94$, $p < .001$, hot, $F(3, 445) = 7.07$, $p < .001$, sunny, $F(3, 445) = 14.81$, $p < .001$, dry $F(3, 445) = 5.41$, $p = .001$, grey/ cloudy, $F(3, 445) = 14.36$, $p < .001$, long day, $F(3, 445) = 11.25$, $p < .001$, high pollen count, $F(3, 445) = 3.32$, $p = .02$, fog/ smog, $F(3, 445) = 7.91$, $p < .001$, and short day, $F(3, 445) = 13.43$, $p < .001$.

Subsequent post hoc t -tests were conducted for pairwise group comparisons in all significant univariate DVs, and p -values are shown in Table 4. Main findings from Table 4 show both SAD groups to feel worse than controls in short daylight, grey/ cloudy days, fog/ smog, and cold weather, and to improve in mood on hot, and sunny days, and long daylight. C-SADs are also worse than S-SADs in cold weather, dry weather, and in short daylight. These differences between the groups for response to weather conditions can be seen from Figure 3.

Summary of results for the reactivity to weather conditions. Group differences were shown in all weather conditions except humid. C-SADs and S-SADs gave more extreme responses than controls, feeling worse in winter conditions for example, short daylight and grey/ cloudy conditions, and improved in

Table 4

LSD Probability Values for all Pairwise Comparisons between Clinical SAD (C-SAD), Subsyndromal SAD (S-SAD), Mixed Seasonal (Mixed), and Control Groups Following Significant Univariate F tests for Weather Conditions (Cold, Hot, Sunny, Dry, Grey and Cloudy, Long Daylight, High Pollen Count, Fog and Smog, and Short Daylight)

Group comparison		Cold	Hot	Sunny	Dry	Grey/Cloudy	Long day	High pollen	Fog/ Smog	Short day
Probability value										
C-SAD	S-SAD	.028	ns.	ns.	.027	ns.	ns.	ns.	ns.	.001
	Mixed	.000	.002	.000	.000	.000	.002	ns.	.012	.000
	Control	.000	.004	.000	.001	.000	.000	.019	.000	.000
S-SAD	Mixed	.000	.000	.01	ns.	.000	ns.	ns.	.025	ns.
	Control	.000	.002	.000	ns.	.000	.000	ns.	.000	.000
Mixed	Control	ns.	ns.	.001	ns.	ns.	.002	.004	.026	.002

mood in long daylight, hot, and sunny days. C-SADs also gave more extreme responses than S-SADs in winter conditions including short daylight.

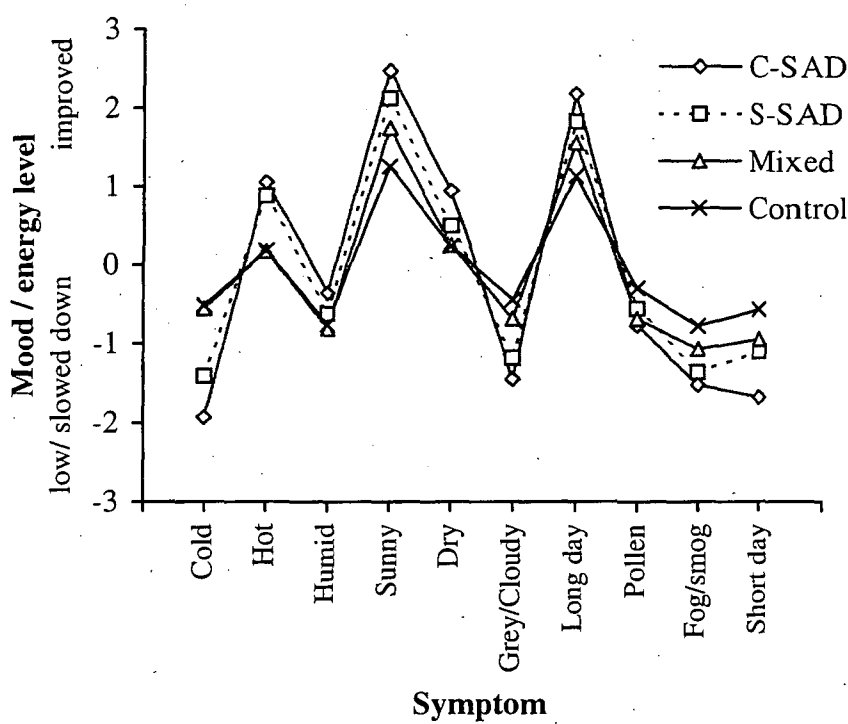


Figure 3. Reactivity to weather conditions as retrospectively rated on the Seasonal Pattern Assessment Questionnaire by clinical SAD (C-SAD), subsyndromal SAD (S-SAD), mixed seasonals, and controls

Hours of sleep per twenty four hour period

A multivariate 4 x 4 (Group x Season) profile analysis MANOVA revealed main effects for season, Pillai's Trace = 0.53, $F(3, 1458) = 183.32, p < .0001$, and for group, $F(3, 486) = 2.73, p = .04$, and a significant interaction between season and group, Pillai's Trace = 0.18, $F(9, 1458) = 10.37, p < .001$. Post hoc analyses revealed C-SADs, S-SADs, and mixed seasonals to report significantly more hours

of sleep in autumn than controls. For winter, significant differences were revealed between all pairwise comparisons at the $p = .001$ significance level, except for the difference between S-SADs and mixed seasonals which was significant at the $p = .01$ level. C-SADs report more hours of sleep than S-SADs, mixed seasonals, and controls, S-SADs sleep more than mixed seasonals and controls, and mixed seasonals sleep more than controls. In summer, the C-SAD group report significantly less hours of sleep than mixed seasonals and controls, and a strong tendency for S-SADs to report less hours of sleep than controls ($p = .052$). No differences between groups are evident for spring.

Repeated measures univariate analyses and LSD post hoc pairwise comparisons for each group separately across the seasons revealed significant differences for all groups. Significant differences were revealed between all season pairwise comparisons for C-SADs, S-SADs, and mixed seasonals with longest hours slept for winter, then autumn, spring, and least hours slept for summer. Controls report sleeping longer hours for winter than for autumn, spring, and summer, at the $p = .001$ significance level, though they did not differ between spring and summer.

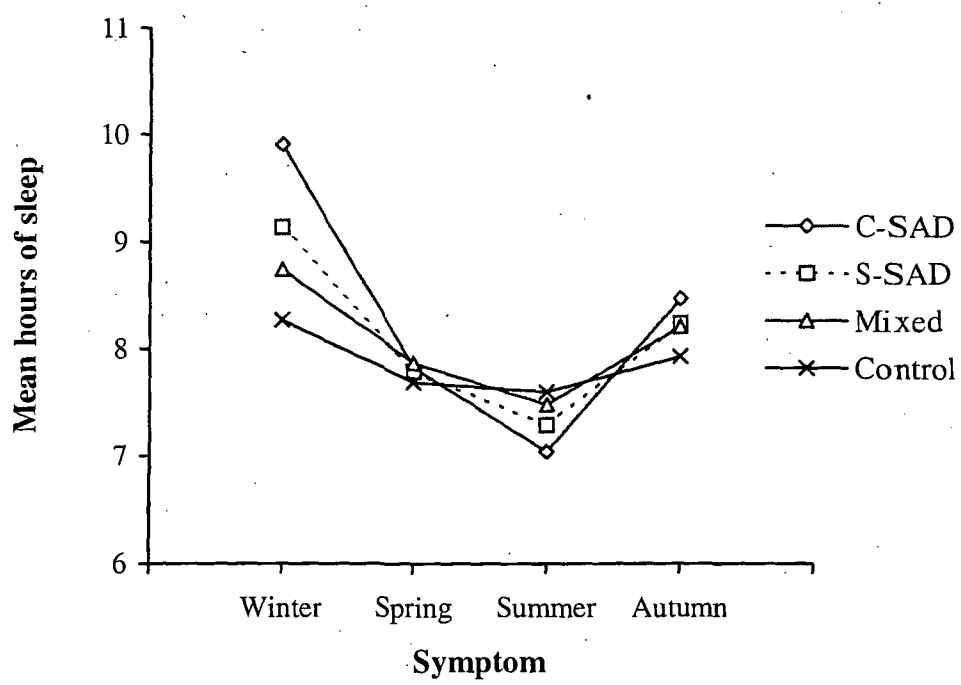


Figure 4. Mean hours of sleep retrospectively self-reported in a 24 hour period for each season for clinical (C-SAD) and subsyndromal (S-SAD) SAD groups, mixed seasonals, and controls

Summary of hours of sleep per season. The groups varied in their hours of sleep across the seasons. The two SAD groups report more sleep in autumn and winter than Controls. For winter, C-SADs also report more sleep than S-SADs. In summer, C-SADs sleep less than controls. All groups sleep longer in winter than in spring, summer, and autumn. The groups did differ in hours of sleep in spring.

Principal components analysis

Principal components extraction with varimax rotation was performed on thirty-five items from the SPAQ. The solution was restricted to three factors after inspection of the scree plot to reduce complexity among factors and to maximise interpretation. These three components together explain 44.5% of the total pooled variance. Loadings of variables on factors, communalities, and percentages of variance are shown in Table 5. Factor loadings under .30 are not shown. Factor 1, a seasonal weather factor, has high loadings on weather variables and months of the year. Winter weather items load positively, and summer items load negatively. Factor 2, a SAD factor, has high loadings on mood and atypical symptoms, SPAQ ratings, and also includes weather variables long daylight, short daylight, and fog / smog. Factor 3 is metabolic with loadings on variables relating to sleep and weight. Three variables, 'weight', 'hours of sleep in winter', and 'sunny' in the solution were complex. Only one item: 'March' did not load on any factor.

Summary. The principal component analysis indicated a three-factor solution: a seasonal weather factor, a SAD factor, and a metabolic factor.

Table 5

Factor Loadings, Communalities (h^2), and Percents of Variance for Principal Components Extraction with Varimax Rotation on Seasonal Pattern Assessment Questionnaire Items

Category	Variable	Factor 1	Factor 2	Factor 3	h^2
Symptom	Appetite		.59		.40
	Energy		.67		.48
	Mood		.73		.57
	Sleep			.48	.29
	Social		.50		.32
	Weight		.54	.31	.41
Weight fluctuation	Weight variation			.46	.25
Hours of sleep in each season:	Winter	-.49		.68	.75
	Spring			.70	.52
	Summer	.47			.31
	Autumn			.79	.73
Reactivity to climate conditions:	Cold	.56			.42
	Dry	.30			.10
	Fog / smog		-.41		.24
	Grey / cloudy	.54			.45
	Hot	-.64			.42
	Humid	-.45			.27
	Long daylight		.50		.27
	High pollen count	-.36			.23
	Short daylight		-.62		.41
	Sunny	.34		.42	.29
Months of Year	January	-.81			.75
	February	-.77			.64
	March				.09
	April	.63			.41
	May	.81			.69
	June	.80			.76
	July	.78	.34		.73
	August	.71	-.35		.64
	September			-.39	.30
	October			-.48	.24
	November		-.43		.29
	December	-.70			.56
	Global seasonality score		.86	.34	.87
	Global rating		.66		.47
Percent of variance (%)		26.5	11.2	6.7	44.5

Discriminant function analysis

A hierarchical discriminant function analysis was performed on the three groups: C-SAD, S-SAD, and controls to determine the best set of predictors for group membership. Three hundred cases were analysed. Of the original 502 cases, the mixed seasonals ($n = 196$) were excluded due to the mixed nature of the group, and their similarity in degree of seasonal change to S-SADs. A further six cases with missing data were excluded.

The first set of predictors was the six symptoms: degree of affect by seasonal change to appetite, energy, mood, sleep, social activity, and weight. One statistically reliable discriminant function was calculated, $\chi^2(12) = 437.77, p < .0001$. A second set of predictors, the number of hours of sleep for each season was added. Finally, GR was added. Thus the groups are separated by symptom ratings, hours of sleep for each season, and GR. Discriminant analysis was reliable in predicting the group membership with 90% of cases correctly classified. Of these, 98% were control, 92% were C-SAD, and 82% were S-SAD.

Summary of results from discriminant function analysis. After exclusion of the mixed seasonal group, reliable classification was made from discriminant function analysis on all three groups, C-SAD, S-SAD, and Controls. Group prediction was reliably made from variables, Symptoms, Hours of Sleep, and GR.

Multiple regression analyses

Multiple regression analyses were conducted using the stepwise method to investigate relationships between the SPAQ variables. The first analyses used the six symptoms as predictors with GSS, and then GR, as the dependent variable. Secondly, hours of sleep for autumn, winter, spring, and summer were used as predictors with the symptom sleep as dependent variable. The summary of the multiple regression analyses appears in Table 6.

For the GSS, all six of the predictors contributed significantly to the DV (see Table 6). The first three predictors were the metabolic variables, energy, weight, and sleep, which contributed 87% of the shared variability, while social activity, mood, and appetite, added a further 13%. For the GR, only four of the six predictors contributed significantly to 30% of the variance. These were firstly mood, then the metabolic variables sleep, energy, and weight (see Table 6). Social activity and appetite did not contribute significantly to prediction of GR. For the second multiple regression analysis, the hours of sleep for each season: autumn, winter, spring, and summer were used as predictors for the dependent variable sleep symptom. Analysis showed that the sleep rating could be predicted from hours of sleep in winter and summer only. Hours of sleep in autumn and spring did not contribute significantly.

Further multiple regression analyses were conducted using the ten weather variables as predictors, with GSS and GR as dependent variables. A summary of the stepwise regression analyses using the weather variables is shown in Table 7. For the GSS, three of the predictors contributed significantly to the variability. These

Table 6

Summary of Stepwise Regression Analyses for Predictors of Global Seasonality Score (GSS) and Global Rating (GR) and Hours of Sleep

DV	Step	Predictors	F	df	B	SE B	β	R ²	R ² change
Analyses using SAD symptoms									
GSS	1	Energy	810.78	492	3.30	0.12	0.79***	0.62	0.62
	2	Weight	934.93	491	1.91	0.10	0.46***	0.79	0.17
	3	Sleep	1044.31	490	1.36	0.08	0.30***	0.87	0.07
	4	Social	1465.50	489	1.10	0.06	0.26***	0.92	0.06
	5	Mood	2843.20	488	1.04	0.04	0.25***	0.97	0.05
	6	Appetite	1.57E+17	487	1.00	0.00	0.25***	1.00	0.03
GR	1	Mood	151.11	492	0.40	0.03	0.48***	0.24	0.24
	2	Sleep	94.58	491	0.20	0.04	0.22***	0.28	0.04
	3	Energy	68.59	490	0.14	0.40	0.16***	0.30	0.02
	4	Weight	53.64	489	0.11	0.41	0.13**	0.31	0.01
Hours of sleep in:									
Sleep	1	winter	53.7	487	0.25	0.03	0.32***	0.10	0.10
	2	summer	42.03	486	-0.18	0.03	-0.23***	0.15	0.14

Note. ** $p < .01$, *** $p < .001$

Table 7

Summary of Stepwise Regression Analyses for Predictors of Global Seasonality Score (GSS), Global Rating (GR) and Metabolic Symptom Score (MSS)

	Step	Predictors	<i>F</i>	<i>df</i>	<i>B</i>	SE <i>B</i>	β	R^2	R^2 change
Analyses using weather conditions									
GSS	1	Short day	34.95	447	-1.13	0.19	-0.27***	0.07	0.07
	2	Sunny	28.45	446	0.72	0.16	0.21***	0.11	0.04
	3	High pollen count	22.31	445	-0.53	0.18	0.13**	0.13	0.02
GR	1	Short day	62.11	447	-0.29	0.04	0.35***	0.12	0.12
	2	High pollen count	34.67	446	-0.09	0.04	0.11*	0.14	0.01
	3	Long day	16.57	445	0.08	0.04	0.11*	0.14	0.01
MSS	1	Sunny	25.51	445	0.44	0.09	0.23***	0.05	0.05
	2	Short day	19.53	444	-0.38	0.11	-0.17***	0.08	0.03
	3	High pollen count	16.29	443	-0.29	0.10	-0.14**	0.10	0.02
	4	Grey and cloudy	13.80	442	-0.26	0.11	-0.12*	0.11	0.01
	5	Humid	12.03	441	-0.17	0.08	-0.10*	0.12	0.01

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

were short daylight, sunny, and high pollen count. For the GR, three of the predictors contributed significantly to the variability. These were short daylight, high pollen count, and long daylight. Because the three metabolic symptoms, energy, sleep, and weight, contributed a high percentage of the variability to GSS, these were added together to produce a metabolic symptom score (MSS) and included as a dependent variable to determine any relationship with the ten weather conditions. Five of the predictors: sunny, short daylight, high pollen count, grey/cloudy days, and humid, contributed significantly to prediction of the MSS.

Summary for predictors of SPAQ scores. Multiple regression showed all six symptoms to contribute to the global seasonality score. The first three were the metabolic symptoms (energy, weight, and sleep) and these contributed a high percentage of the shared variance. Mood, followed by energy, weight, and sleep, predicted the overall rating of participants' perception of seasonal disturbance in their lives. Further multiple regression analyses found short daylight to emerge as a consistent predictor of global seasonality score, global rating, and a metabolic symptom score.

Discussion

Seasonal variation is evident in Tasmania with up to seventy six percent of the sample recording changes to mood and/ or associated atypical vegetative behavioural symptoms across the twelve-month period. Of these, up to nine percent record symptoms at a clinical level of severity, and twenty six percent record

symptoms at a subsyndromal level of severity, according to Rosenthal et al.'s (1993) criteria for diagnosis. Confirmation of the recurrent seasonal nature of depressive episodes for the previous two years as well as the absence of external factors, are also required for a diagnosis of SAD. These figures are comparable with the high level of seasonal variation to atypical symptoms recorded from Maryland (Rosenthal et al., 1984) and New York (Kasper et al., 1989). All participants regardless of group reported sleeping longer in winter than in autumn, spring, or summer, further confirming the high level of seasonality in the general population. The current study has shown that up to 9% may experience SAD at a clinical level, consistent with previous findings that vary from 3% to 12%. In contrast, 24% report seasonal change at a subsyndromal level, compared to previous findings varying from 13 – 18%. This higher prevalence of S-SAD may reflect the disruptive effects of the Tasmanian winter on lifestyle and social activities, which may be more pronounced in the university student sample. In the northern hemisphere, Christmas falls in mid-winter reflecting conservative prevalence rates due to the festive spirit and likelihood of greater socialisation at this time of year.

Regardless of group, the seasonal profiles or pattern of seasonality show all participants fluctuate in mood and behavioural symptoms to some degree. However both SAD groups show greater fluctuations between summer and winter than controls, with C-SADs being more extreme than S-SADs. The seasonal profiles show all groups to feel worst, sleep most, eat most and gain weight, and socialise least, during the winter months. The opposite pattern: feeling best, sleeping least, eating least, losing weight, and socialising most, is evident in summer in all groups from the profiles of symptoms.

Analyses using ANOVA and MANOVA differentiated the two SAD groups from controls for all symptoms. The SAD groups experience a greater degree of seasonal change to sleep, social activity, mood, appetite, weight, and energy than non-seasonal controls, with C-SADs giving more extreme responses than S-SADs, and thus validating group selection. These differences were also repeated for GR, WTFluct, and hours of sleep per season. The groups also differentiated in their reactivity to weather conditions, with the SAD groups feeling worst for short daylight, grey/ cloudy days, and fog/ smog, and feeling improved in mood for long daylight and sunny days.

After exclusion of the non-SAD mixed seasonal group, a discriminant analysis shows the SPAQ to be reliable in classifying high percentages of the remaining three groups; firstly by the symptom ratings, then by the hours of sleep in each season, then global rating.

Three factors emerged from a factor analysis. These were labelled as a Seasonal Weather factor, a SAD factor, and a Metabolic factor. Madden, Heath, Rosenthal, & Martin (1996) conducted a factor analysis using participants from varying latitudes across Australia, from the seasonal profile items for each month. Their factor structure showed both winter and summer factors. In contrast, the current study showed no participant to have their depressive episodes during the summer months. However the results show support for Madden et al.'s two winter

factors with one factor loading highly on mood, and the second factor loading highly on the atypical vegetative symptoms.

Multiple regression analyses were conducted to determine relationships among the variables. Whilst all symptoms were predictors of GSS, the metabolic symptoms, energy, weight, and sleep, contributed to a high level of the variance with social activity, mood, and appetite only adding an additional thirteen percent. Mood was found to be the best predictor of the GR, followed by the metabolic symptoms, sleep, energy, and weight. The short daylight weather condition consistently emerged as a predictor of GSS, GR, and MSS, further affirming the importance of the photoperiod as an aetiology in SAD.

Several findings of importance in understanding SAD have emerged from the study.

Continuum of severity

The study aimed to seek any evidence for SAD being an extreme of seasonality in the general population. The pattern of severity in the seasonal change of symptoms, together with the seasonal symptom profiles, suggest that SAD may be an extreme of seasonality in the general population. Further, the high contribution of metabolic symptoms, energy, sleep, and weight, to the variance in GSS gives support to SAD being an extreme of the seasonal change and/ or disturbance evident in the normal population.

On the other hand, the high reliability of group membership from discriminant analysis suggests a distinction between groups. Further distinctions from the analyses are shown. For example, C-SADs feel greater disturbances from all symptoms, rate GR higher, and show a greater seasonal weight fluctuation, than S-SADs. C-SADs also report more hours of sleep in winter, and less in summer, than S-SADs. However from the reactivity to ten weather conditions, the two SAD groups only differ from each other for short daylight, cold, and dry days, with C-SADs feeling worse for short daylight and cold days, and feeling improved in mood on dry days, as compared to S-SADs.

Any apparent continuum of severity may be due to the lineal nature of the measurement of the GSS range. Further investigation is required to clarify the relationship between SAD and seasonality in the general population.

Hypomania during spring/ summer

The seasonal profiles confirm the prevalence of the symptoms in winter for the two SAD groups and also show this tendency for all groups to some degree. The profiles support bi-polarity for all symptoms. Increases in mood and socialising, together with decreases in weight, eating, and sleep during the summer months from the profiles indicates hypomania in the Tasmanian sample. Compared to controls, both SAD groups sleep significantly longer hours for the autumn and winter months and significantly less hours for summer, offering further support for hypomania. Bi-polarity is also suggested from the factor analysis with winter and summer items loading positively and negatively on a Seasonal Weather factor. Others have found

it difficult to diagnose hypomania. Any improvement in mood and behavioural symptoms may be merely relief from winter symptoms. Further research using a longer time base (e.g., twelve months) is required to confirm the presence of hypomania.

Sleep and Metabolic Symptoms

Previous findings have suggested that the metabolic cluster of SAD symptoms, energy, weight, and sleep, may serve as an energy serving function in SAD (e.g., Putilov, 1998; Skwerer et al., 1989). The current study adds further support to the importance of the metabolic symptoms, energy, sleep, and weight, in seasonal disturbance and/ or in SAD. This metabolic emphasis may indicate similarities with hibernation in animals, and may have implications for further understanding the nature of SAD.

Limitations

A limitation of the study is that recent findings (Thompson & Cowan, 2001) suggest that the SPAQ does not have good face validity for C-SADs, and may overestimate their prevalence. However, the SPAQ is a good screening questionnaire and may be safely used with additional information required to satisfy criteria prior to any experimental procedure. Concerns with the accuracy of retrospective self-rating have been noted. Participants in the current study were not informed of any specificity to a season prior to completion of the SPAQ in the current study, therefore no seasonal cognitive bias may be expected. Previous

studies have gained participants by mail/ telephone surveys, with completion by a proportion of questionnaire recipients only. The present study had virtually 100% compliance by participants attending the particular laboratory session.

All participants from the present study were university students, indicating that the majority were young adults of above average intelligence and therefore may not represent the general population. Replication of the survey using a more representative sample of the general population is required to confirm the external validity of the findings.

Conclusions

A high percentage of the general population experience seasonal variation to mood and associated behavioural symptoms. Decreases in mood, energy, and social activity; and increases in eating, weight, and sleep were reported for the autumn/ winter months. Of these, up to nine and twenty six percent may experience SAD at clinical and subsyndromal levels respectively, though further information is required for a diagnosis of SAD to confirm these estimates. Evidence was sought to determine whether SAD is an exaggeration of seasonal disturbance in the general population. However, controversial findings from this study suggest any apparent continuum of severity may be due to the lineal nature of the measurement of seasonal disturbance and additional research is required to confirm the nature of SAD in relation to seasonality. For example, the study shows the importance of the metabolic symptoms in SAD. This finding supports the dual vulnerability hypothesis (Young, Watel, Lahmeyer, & Eastman, 1991) which proposes that SAD

sufferers experience initial seasonal disturbance to the atypical vegetative symptoms, followed by depressive episodes in vulnerable individuals.

Increases in mood, energy, and social activity; and decreases in sleep, eating, and weight were evident in the SAD groups during the spring/ summer. The study offers support for hypomania in SAD during the spring/ summer months in the Tasmanian sample, rather than just a remission of symptoms.

Further confirmation for the photoperiod as an aetiology in SAD was obtained, with the climatic condition 'short daylight' consistently emerging as a predictor of participants' global seasonality score, their overall perception of seasonal disturbance of SAD in their lives, and a metabolic symptom score.

Overall, the study has shown that up to seventy six percent of the Tasmanian sample experience seasonal variation to mood, and associated atypical vegetative symptoms. Of these, up to nine percent may have SAD at a clinical level, and twenty six percent at a subsyndromal level. The SPAQ differentiated between the groups on degree of seasonal disturbance to all symptoms, thus validating the grouping methodology. A high percentage of the SAD groups as well as controls were correctly classified in a discriminant analysis, further validating group selection. The SAD groups reported hypomania for the spring/ summer months.

The results indicate the importance of the metabolic symptoms. For example, a three-factor structure emerged from a factor analysis giving a Seasonal Weather factor, a SAD factor, and a Metabolic factor. Further, the metabolic

symptoms, energy, weight, and sleep contributed to a high percentage of the shared variance in multiple regression analyses. The climatic condition 'short daylight' consistently emerged as a predictor of the global seasonality score and also in participants' overall perception of SAD as a problem in their lives, further confirming the shorter daylight hours in winter as an aetiology in SAD.

Further research to be conducted across the twelve-month period will add to the current findings. Two longitudinal studies (chapters 7 and 8) will enable a further understanding of any linearity in seasonality and whether SAD is an extreme of this seasonality. The longitudinal studies will also seek evidence for SAD being a disorder distinct from NSD.

Chapter 5

Seasonal Affective Disorder and Therapies

Chapter 5: Seasonal Affective Disorder and Therapies

*"Ah, woe is me! Winter is come and gone,
But grief returns with the revolving year."*

Percy Bysshe Shelley

Whilst seasonality in affective illness has been documented throughout history (see Chapter 3), seasonal affective disorder (SAD) has only been proposed as a disorder since 1984 after Rosenthal and his co-workers commenced research in this area at the National Institute of Mental Health at Maryland, US. The clinical features of SAD will be described in this chapter, and a comparison made with non-seasonal major depressive disorder (NSD). Light therapy is effective in reversing the depression and associated atypical behavioural symptoms in SAD, however the mechanism of action in light therapy remains unclear. Alternative therapies include drugs, psychological approaches, and lifestyle changes.

SAD: The clinical picture

Sufferers of SAD experience recurrent depressive episodes reliably each year during the autumn/ winter months along with atypical vegetative symptoms, hypersomnia, overeating with carbohydrate craving, weight gain, and lack of energy. Additional problems include fatigue, social withdrawal, and decreased libido (Rosenthal et al., 1984). During the spring/ summer months, sufferers report either, a remission of symptoms, or hypomania. Whilst similarities with bipolar disorder have been noted, the hypomania or mild upswings in mood in SAD, are

noticeable by others, though not severe enough to markedly impair day to day functioning (American Psychiatric Association, 1994).

SAD sufferers experience disruptions to their daily living patterns during winter. Some report difficulty in holding employment and relationship problems. Performance difficulties in the workplace may include, for example, motivational deficits, decreased productivity, inability to concentrate, difficulty in making decisions, or difficulty in initiating new projects (Rosenthal, 1993). Disruptions to cognitive patterns include psychomotor retardation, memory problems (Healy, 1987), and visuo-spatial deficits (Michalon et al., 1997; O'Brien et al., 1993). Depressed mood ideation in SAD also includes anxiety, guilt, low self-esteem, and sadness (Terman et al., 1989).

A further group of people experience the atypical vegetative SAD symptoms reliably during autumn/ winter, though with a lesser degree of mood variation. Known as subsyndromal SAD (S-SAD) or “winter blues”, this syndrome has also been referred to as “seasonal anergy syndrome” in Switzerland (Wirz-Justice et al., 1989) and “winter complainers” in New York (Terman, 1989). S-SAD sufferers frequently experience a greater degree of seasonal variation to the behavioural symptoms than to mood (Kasper et al., 1989; Terman). Thus it has been suggested that this cluster of SAD symptoms serve an energy conserving function through the autumn/ winter months. S-SADs are generally able to cope through the winter by making their own adjustments to daily living, with clinical intervention generally not required.

Epidemiological research conducted in Hobart, Tasmania, (refer Chapter 4) found up to nine percent may experience mood and behavioural disturbances at the clinical level and twenty six percent at the subsyndromal level. This finding suggests that a percentage of the population experiencing SAD are unable to cope efficiently with daily living through the winter months. As yet SAD has not been validated as a disorder distinct from major depressive disorder. The DSM IV (American Psychiatric Association, 1994) includes a Seasonal Pattern Specifier which may applied to recurrent major depressive disorder. Currently there is no medical test to confirm SAD and diagnosis is based on history alone. A further difficulty remains in determining what constitutes a clear-cut distinction between C-SAD and S-SAD.

Differences between SAD and non-seasonal depression. SAD differs from NSD in both symptoms and physiology indicating that SAD is a disorder distinct from major depressive disorder. The atypical vegetative symptoms of SAD, hypersomnia, overeating, and weight gain are opposite to the typical vegetative symptoms most likely to be experienced with non-seasonal Major Depressive Disorder (NSD): the symptoms typical of NSD are insomnia, under-eating, and weight loss (American Psychiatric Association, 1994). A considerable amount of the literature confirms that SAD is biologically based (see chapter 6), with depressive episodes having an onset in autumn with the decrease in the amount of daylight hours, and hypomania or remission during spring and summer. Psychosocial stressors and unpleasant winter anniversaries are excluded as

diagnoses in SAD. In contrast, the origin of NSD may be either exogenous relating to external stressors, or endogenous with disturbances to biochemical processes.

Light therapy is specific to seasonal depression (Rosenthal et al., 1989). The success of light therapy is due to its suppressing effect on melatonin. In non-seasonal depression, melatonin levels are low during winter (in contrast to high melatonin levels during winter in SAD) (Shafi & Shafi, 1990), hence any attempt of light therapy would worsen the depression. Previous research by Austen and Wilson (2001) showed hypoarousal in S-SAD during winter, with an increase in vagal tone and a decrease in heart rate, representing high parasympathetic and/ or low sympathetic arousal. On the other hand, hyper-arousal (Lam & Levitan, 2000) and low vagal tone (Balogh et al., 1993; Rechlin et al., 1994; Rechlin et al., 1995) have been noted in NSD.

A summary of the SAD profile shows C-SAD and S-SAD sufferers to experience depressive episodes and associated atypical vegetative symptoms recurring reliably each year during the autumn and winter months. Additional behavioural, performance, and cognitive impairments create further problems for the SAD sufferers' daily living pattern. SAD differs from NSD in both symptoms and physiology suggesting that underlying mechanisms in SAD differ from those in NSD.

Light therapy

Light therapy as a treatment for SAD began after an anecdotal comment by Lewy in 1980 that sunlight could suppress melatonin production. Lewy and his co-workers were the first to report the use of bright light therapy to treat a patient with seasonal pattern in recurrent mood swings (Lewy, Sack, & Singer, 1990; Rosenthal, 2000). The patient became depressed as the days shortened in the winter, with the depression lifting as the days lengthened in the spring. They found they were able to lift his depression after four days of exposure to 2000-lux light from 6 to 9 a.m. and from 4 to 7 p.m., to extend day length by simulating a summer day.

The biological pathway for circadian entrainment of light is the retino-hypothalamic tract, a pathway extending from the retina to the SCN (Hill, 1992). The therapeutic effect of light is thought to be a suppression of excess melatonin secretion by the resetting of circadian rhythms in the SCN. A large body of research has been conducted on light therapy that may be divided into two categories. Firstly, experimental research has been conducted to determine the most efficient method of administering the therapy, with studies varying colour and intensity of light, duration of presentation, time of day, and method of presentation. Secondly, a full understanding of the therapeutic effect of light is not yet known, and experimentation has been conducted to determine the mechanism of action for light therapy that may help in determining the pathogenesis in SAD. Several theories have been developed from the successful therapeutic effect of light and will be described in Chapter 6 in an endeavour to gain a greater understanding of the pathophysiology in SAD and the mechanism of action in light therapy.

Efficacy of light therapy in SAD. Light therapy began with a large cumbersome metal box, containing full spectrum fluorescent lights and covered by a diffusing screen, later being updated to a smaller light box model (Rosenthal, 1993). A newer portable light visor was developed to be worn like a cap and has light suspended above and in front of the eyes. Advantages of the visor are that the wearer may move around while receiving treatment, and the intensity of light varied to suit the individual. Levitt, Joffe, and King (1993) and Teicher et al. (1995) have suggested that the light visor's success may be a placebo effect. An alternative possibility is that the regular wearing of the visor may function as a time cue to reset the circadian clock (Teicher et al.).

Experimentation with varying intensities of light have found bright light at 2500 lux for two hours each day for a period of at least one week to be the most effective. Whilst a higher intensity of light at 10,000 lux for thirty minutes is effective, a high relapse rate on discontinuation has been shown (Terman et al., 1988). Consistent daily exposure to light therapy is required during the high-risk period to maintain remission of symptoms. The amount of light required varies among individuals and also depends on the intensity and duration of environmental light available. Presentation of light to the eye is superior than to presentation to the skin (Wehr, Skwerer, Jacobsen, Sack, & Rosenthal, 1987) that may be due to the ability of receptors in the retinal periphery to take in light.

The time of day for therapy has also been considered in the efficacy of light therapy in SAD. A large body of research has been conducted with mixed findings. The assumption for morning therapy is based on the circadian phase delay (Lewy et al., 1989). Some researchers maintain treatment is more effective when bright light is scheduled in the morning (e.g., Lewy et al., 1990; Wirz-Justice et al., 1989), while others prefer evening light (e.g., Helleckson, 1989). Many sufferers have found that light therapy is successful in remission of symptoms regardless of the time of day for administration (Rosenthal, 1993). It is possible that the optimal time of day for light therapy differs among individuals.

Dawn simulation, an alternative to light therapy was developed by Terman et al. (1989) as a convenient, lower cost treatment available for milder symptoms and has been shown effective in SAD (Avery et al., 2001; Norden & Avery, 1993). The simulator is programmed to turn on at a pre-determined time, with the intensity of light increasing gradually creating an artificial dawn during the winter months.

The possibility of a placebo effect has been considered and experiments conducted (Teicher et al., 1995) in an attempt to eliminate this possibility. Placebo conditions have included varying intensities (e.g., Rosenthal, 1984;) and colours (Oren, 1991) of light, administration at varying times of day (e.g., Eastman, Young, Fogg, Liu, & Meaden, 1998), or drugs (e.g., Neumeister, Praschak-Rieder et al., 1998; Neumeister, Turner et al., 1998) using double blind crossover conditions. Overall, placebo studies have shown remission to a degree, however more research is required to confirm the findings. Due to the nature of light therapy, a difficulty

remains in conducting a double-blind study with a conventional placebo. The possibility exists that light therapy involves a regulation of lifestyle that strengthens environmental rhythms, thus resetting circadian rhythms (Healy & Waterhouse, 1995). It has also been considered that both patient expectations (Eastman et al.) and/ or the pre-treatment SAD patient/ therapist relationship may have a positive influence on any outcome from light therapy (Geerts, Kouwert, Bouhuys, Meesters & Jansen, 2000) making any attempt at elimination of placebo effect difficult.

Side effects of light therapy. Due to the strong effect of light on the eye, the possibility of damage to the eye and/ or visual pathway has been examined. Early studies (e.g., Rosenthal et al., 1984; Rosenthal, Kasper, Schultz, & Wehr, 1989) reported mild headaches, irritability, and nausea. More recent studies (e.g., Levitt et al., 1994) report eyestrain and the feeling of being “wired”, dryness of eyes, dryness of nasal passages. Kogan and Guilford (1998), with a shorter exposure and stronger intensity at 10,000 lux, found 20% of their sample to experience mild headaches and/ or visual problems. After extensive ophthalmological examinations in SAD patients, Gallin et al. (1995) found no ocular abnormalities either before or after both short term and long term (up to six years) therapy. In some cases, light therapy has been known to initiate a state of overactivity along with insomnia and irritability (Rosenthal et al., 1989). Light therapy can be cumbersome and time consuming, and continuing treatment is required for success. Any side effects present need to be weighted against improvement in mood and remission of symptoms.

Overall, light therapy is successful in reversing the depressive episodes and associated behavioural symptoms in SAD, both at clinical and subsyndromal levels. Studies have been conducted to determine the most efficient method of its administration. Gaining an understanding of any mechanism involved in the reversal of symptoms may also assist in understanding the pathogenesis of SAD. Some side effects have been noted, though these are mild and need to be weighted against the benefits of the reversal of symptoms in SAD. Light therapy is specific to SAD, which may help any distinction from NSD.

Understanding the mechanism of light therapy. A review of the experimental literature on light therapy indicates the successful reversal of the symptoms in SAD, and has also shown the reversal of the pathophysiological abnormalities to normal using pre- and post-therapy experimental designs. Several abnormalities have been noted in the pathophysiology of SAD, and will be described in chapter 6.

Other therapies

Whilst light therapy is the accepted form of treatment for SAD, up to forty percent of patients do not respond and require an alternative or additional treatment. Alternative treatments to light therapy have included drugs, psychological therapies, and lifestyle changes.

Drugs. The range of selective serotonin re-uptake inhibitor (SSRI) drugs has been effective in the treatment of SAD (Hilger et al., 2001; Rosenthal, 1993).

Because serotonin levels are low during winter in SAD, SSRIs enable an increase that is thought to be helpful in reversing the symptoms. SSRIs have few side effects, though some have reported nausea, irritability, anxiety, and sedation. Because of the seasonal nature of SAD, it is necessary to vary the dose according to seasons.

Noradrenergic pathways are involved in SAD (e.g., Shafi & Shafi, 1990; Skwerer et al., 1989) and recent findings report the effectiveness of drugs based on norepinephrine. For example, Reboxetine (Hilger et al.), a selective noradrenaline re-uptake inhibitor and Mirtazapine (Hesselmann et al., 1999) enables the re-uptake of both noradrenaline and serotonin. These findings support suggestions of involvement of catecholamine pathways in the pathophysiology in SAD.

Psychological approaches. Because depressed mood ideation in SAD includes negative thought patterns, cognitive dysfunction, anxiety, and guilt, similar to those in NSD, it has been suggested that psychological approaches to therapy, for example cognitive behavioural therapy, may be successful in SAD (Hodges & Marks, 1998; Rosenthal, 1993). Hodges and Marks found greater negative automatic thoughts and depression in both SAD and NSD as compared to controls: the two groups did not differ from each other on either measure. Cognitive behavioural therapy deals with behavioural problems as well as cognitive distortions. The therapist determines an individual approach with an emphasis on each SAD patient taking responsibility for his/ her own behaviour modification.

Lifestyle changes. A self-help book, Winter Blues (Rosenthal, 1993) was written to assist SAD sufferers to understand their disorder. Lifestyle changes that may assist SAD sufferers include adaptations to their environment, diet, and

undertaking exercise. SAD sufferers generally recognise that their problems relate to the short daylight in winter, and helpful adaptations include maintaining adequate natural light, sunshine, and warmth. More extreme measures include a change of environment, for example, a holiday, or relocation to a warmer climate.

Due to the tendency for SAD sufferers to eat more and hence gain weight during winter, consideration of diet and exercise is important. A high carbohydrate diet, with reduced calorie intake is recommended, avoiding the tendency to snack on sweets. A diet high in complex carbohydrates, for example, legumes, pasta, potatoes, and grains, may also increase energy level (Rosenthal, 1993). Consumption of carbohydrates may assist in understanding biochemical abnormalities. For example, Wurtman (1990) has shown that by increasing carbohydrate intake, serotonin levels are also increased. Lacoste and Wirz-Justice (1989) found SADs to consume more carbohydrates during winter as compared to autumn, spring, and summer, and also compared to controls. Levels of protein consumed did not differ either across the year, or between groups.

Stress management, for example, avoiding major stresses and/ or other major changes such as moving house or changing jobs during winter, may assist in maintaining control of symptoms in SAD. (Rosenthal, 1993). Determining ways to conserve energy, and advance preparations for winter have also been helpful. Overall, an awareness of individual seasonal changes and an adaptation to individual circumstances can be helpful in minimising stress. Maintaining a regular routine, including restricting hours of sleep through winter, and gaining mid-day sun assist in minimising the depressive episodes in SAD. During the spring and

summer months when energy levels are high, many SAD sufferers use their creativity to advantage. Creative SAD sufferers, for example, musicians Handel and Mahler used the summer period for composing music. On the other hand, a caution not to overcommit during the hypomanic phase is important.

Whilst the success of light therapy is well regarded, other treatments have included drug therapy, cognitive therapy, and behavioural therapy. Lifestyle changes and stress management techniques also assist in SAD. A combination of therapies may be successful for some individuals.

Conclusions

The depressive episodes in SAD, along with associated atypical vegetative symptoms, hypersomnia, overeating, weight gain, social withdrawal, and loss of energy, recur reliably during the autumn and winter months, and hypomania/remission in spring and summer. Additional symptoms include memory difficulties, inability to concentrate, and motivational deficits, and relationship problems can create severe problems for the SAD sufferer. S-SADs, who experience the symptoms reliably in winter, with a lesser degree of mood variation, are generally able to cope by making adjustments to daily living.

The atypical vegetative symptoms of SAD differ from those typical in NSD. Further, several physiological differences, and the specificity of light therapy to

SAD add further support to the contention that underlying mechanisms in SAD differ from those in NSD.

Light therapy is successful in reversing the symptoms of SAD and much research has been conducted to determine the most efficient method of administration. Approaches to determine the mechanism of action involved in light therapy may also assist in understanding underlying mechanisms in SAD and will be described further in Chapter 6. Adaptation to the lack of environmental light in winter by gaining warmth and light, together with lifestyle changes, for example, diet, exercise and stress management assists SAD sufferers. Drugs, cognitive therapy and behavioural therapy have been also shown to assist in controlling the symptoms.

Chapter 6

Theories of Seasonal Affective Disorder

Chapter 6: Theories of Seasonal Affective Disorder

Light therapy is effective in reversing the depression and associated atypical behavioural symptoms in SAD, however the mechanism of action in light therapy is unclear. A large body of experimental research examines the mechanism of action in light therapy using pre- and post- therapy design experiments. In an endeavour further to understand SAD, its aetiology and pathophysiology, and the mechanism of action in light therapy, a review of the theories of SAD will be outlined. Evidence from remission of symptoms due to anti-depressants may also assist in understanding SAD. Due to symptom similarities between SAD and hibernation, and the growing body of research that suggests that SAD may have hibernation-like features, this comparison will also be presented.

Aetiology

To date an aetiology in SAD is unclear, though it is generally thought to be the reduction in environmental light with the reduced daily photoperiod during the autumn/ winter months in locations of greater than 40° latitude (Rosenthal & Wehr, 1992). Short daylight emerged as a consistent predictor of a global seasonality score, participants' overall perception of SAD as a problem, as well as the metabolic cluster of SAD symptoms in Chapter 4 of this thesis.

Considerable evidence exists to suggest that circadian rhythms are disrupted in SAD sufferers in a variety of physiological and psychological responses (Healy, 1987; Hill, 1992). However the reasons for any underlying

vulnerability to the reduced amount of light during the autumn/ winter months is unclear. The recent finding of a biochemical change in melatonin that may signal the change of seasons is similar to the signal found in hibernating animals (Wehr et al., 2001) and it is important because (a) it may implicate vulnerability and (b) gives a further similarity with hibernation.

Biochemical abnormalities have also been shown which implicate the three major neurotransmitters, serotonin, dopamine, and norepinephrine. However, whilst a large body of research investigating neurotransmitters has been conducted, the role that they play in SAD remains unclear. Recent studies (e.g., Austen & Wilson, 2001; Putilov & Danilenko, 1998) implicate ANS involvement with the suggestion that the atypical vegetative symptoms in SAD represent a state of hypoarousal which may serve an energy conserving function.

Theories in seasonal affective disorder

Several theories have been proposed in the pathogenesis of SAD, with a large body of experimental research having been undertaken over the past two decades to assist in further understanding the pathophysiology of SAD, and why some individuals are vulnerable. A large proportion of the research uses light therapy in experiments with pre- and post- light therapy designs. Whilst earlier papers have suggested more specific theories, more recent literature now suggests that a combination of pathophysiologies may be the key to understanding SAD.

Melatonin hypothesis. Melatonin production is influenced by the 24 hour light-dark cycle, with secretion stimulated during darkness, and suppressed with light (Shafi & Shafi, 1990). Hence, with longer hours of darkness during winter, an excess of melatonin is produced. Studies show that on remission of symptoms by light therapy, melatonin secretion is also suppressed (e.g., Shafi & Shafi). However, whilst melatonin is known to have an involvement, its central role in SD has been questioned. Rosenthal and Wehr (1992) conclude after conducting several experiments that “the evidence, taken as a whole, suggests that melatonin secretion does not play a critical role in the pathogenesis of SAD, nor does its modification appear to be the critical element in the mechanism of light action in light therapy” (p. 56).

Circadian theories. Circadian rhythms are disrupted in SAD, though the nature of the disruption is not well understood, and studies give mixed findings. The nature of the disruption was first thought to be a phase delay in circadian rhythms in SAD sufferers due to their tendency to sleep longer in winter. Initial suggestions were that phase advancing of the delayed rhythms by light therapy in the morning would be more effective than in the evening. Whilst some researchers found support for greater morning effectiveness (e.g., Lewy et al., 1998), others found better results at other times of day (e.g., Helleckson, 1989; Jacobsen, Wehr, Skwerer, Sack, & Rosenthal, 1987; Wirz-Justice et al., 1989). The phase shift hypothesis remains controversial. The theory explains the effectiveness of morning therapy, however evening treatment would further delay the rhythm with the possibility of being ineffective or even increasing SAD symptoms (Rosenthal & Wehr, 1992).

An alternative circadian theory suggests that, rather than a phase delay, the overall amplitude of circadian rhythms could be reduced (Czeisler et al., 1989; Rosenthal & Wehr, 1992). It is possible that light exposure might have the effect of phase-shifting circadian rhythms as well as increasing their amplitude: though this theory remains speculative with little research conducted.

Theories involving visual pathways. The retina has been of interest in determining the pathophysiology in SAD, with the suggestion that changes in sensitivity to light may be a factor in SAD. Light is mediated through the retina and travels through the retino-hypothalamic pathway via the suprachiasmatic nucleus (SCN). Early studies suggested the possibility that SAD sufferers differed in their retinal sensitivity to light. However studies show mixed findings with either super-sensitive (Beersma, 1990) or sub-sensitive findings (Lam, Beattie, Buchanan, Remick, & Athanasis, 1991; Remé, Terman, & Wirz-Justice, 1990). Others have shown no differences (e.g., Oren, 1991). A more recent study, Oren et al. (2000) showed the successful remission of SAD symptoms with light therapy in visually impaired SAD participants indicating that light still enters the retino-hypothalamic pathways independently of retinal photo-receptors. The successful presentation of light to the retina as compared to the skin (Wehr et al., 1987) gives additional evidence for an involvement of the visual pathway in the pathophysiology of SAD. Further, visuo-spatial problems for winter that do not remit for summer (Michalon et al., 1997; O'Brien et al., 1993) suggest an abnormality may be present in the visual pathway of individuals with SAD. The nature of this abnormality is unclear. Serotonin is a precursor of melatonin in the retino-hypothalamic pathway. Because of excess levels of melatonin during the winter, its suppressing effect on serotonin

is greater, creating biochemical abnormalities. The SCN, the locus of circadian rhythms, is also included in this pathway.

Neurotransmitter abnormalities. The three major neurotransmitters, serotonin, and catecholamines, dopamine and norepinephrine, have all been implicated in SAD.

Serotonin has been widely investigated in the pathogenesis of non-seasonal mood disorders (e.g., Wurtman, 1990) and has also been implicated in SAD (e.g., Hill, 1992; Shafi & Shafi, 1990). Hypothalamic concentrations of serotonin vary with the seasons with lowest levels during winter, and increases have been shown with remission of symptoms in light therapy (e.g., Jacobsen, Murphy, & Rosenthal, 1989) and drug administration (e.g., Neumeister, Praschak-Rieder et al., 1998; Neumeister, Turner et al., 1998; Reclin et al., 1995).

Dopaminergic systems are abnormal in SAD (Depue, Arbisi, Spoont, Leon, & Ainsworth, 1989; Oren, 1991) suggesting that dopamine might play a role. Depue et al. reported dopamine functioning levels to be lowest in winter, and highest in summer, which may represent decreased and increased states of behavioural activity (e.g., social activity, achievement related patterns, and sexual activity) in winter and summer respectively. Disruptions to noradrenergic pathways (Shafi & Shafi, 1990) may implicate norepinephrine in the pathogenesis of SAD. Lower levels of norepinephrine have been shown in SAD sufferers as compared to controls (Hill, 1992; Shafi & Shafi; Skwerer et al., 1989), returning to normal with

light therapy. Skwerer et al. found resting noradrenergic levels in depressed SAD patients prior to therapy were inversely related to their level of depression. After therapy, resting noradrenaline levels increased in direct proportion to mood. Further in SAD, treatment with β -blockers has shown either no improvement (Hill, 1992) or a worsening (Schlager, 1994) of symptoms.

Autonomic Nervous System involvement. Neurotransmitter disturbances (e.g., disrupted noradrenergic pathways) in SAD sufferers, together with symptoms, such as sleep, energy, and weight, suggest the ANS and/ or metabolism may have an involvement in the pathogenesis of SAD. SAD sufferers sleep longer in the autumn/ winter months, though the extra sleep is not refreshing. Sufferers also report fatigue. A preliminary study conducted by Austen & Wilson (2001) to determine the extent of autonomic involvement found an increase in respiratory sinus arrhythmia (RSA) and a decrease in heart rate (HR) representing increased vagal tone during winter in S-SAD implicating increased parasympathetic, or decreased sympathetic tone. Lam & Levitan (2000) note that hypo-arousal is specific to SAD, while hyper-arousal is characteristic of NSD. Further research to extend and clarify the findings will be outlined in Chapter 7 of this thesis.

Recent literature suggests that more than one mechanism may be involved in the pathophysiology of SAD. For example, Young et al., (1991) propose a dual vulnerability hypothesis that suggests initial seasonal disturbances to sleep and energy, followed by depression in vulnerable individuals. This finding may have important implications determining a clear-cut distinction between clinical and sub-syndromal levels of SAD. It is possible that all SAD sufferers

experience disturbances to the atypical vegetative symptoms, while only those who are vulnerable develop additional depressive episodes. Findings from analyses in Chapter 4 of this thesis may be seen as consistent with the dual vulnerability hypothesis. For example, a factor analysis gave separate factors for metabolic symptoms and for the SAD/ mood factor. Regression analyses also showed predictors, energy, sleep, and appetite, to contribute to a high percentage of the global symptom score, suggesting the high involvement of seasonality in the metabolic symptoms. On the other hand, mood emerged as the best predictor of participants' overall perception of SAD as a problem.

Some researchers (e.g., Putilov & Danilenko, 1998) suggest that light therapy produces an energising effect that activates the sympatho-adrenal system as well as regulation of sleep patterns and circadian rhythms. Putilov and Danilenko believe that any simple pathophysiological theory of SAD is inadequate due to the evidence for several mechanisms that appear to contribute to the remission of symptoms in SAD. They argue that the combined effects of circadian rhythms, sleep regulation, energy regulation, and the sympatho-adrenal systems are all necessary for the success in light therapy, and therefore are an important consideration in any explanation of SAD.

A summary of the literature involving SAD theories and pathophysiology indicates disruption to neurotransmitter production and circadian rhythms that may be due to their close relationship in the retino-hypothalamic pathway and the SCN. Until recently, a physiological marker that triggers winter depression was unknown, though the recent finding of a photoperiodic signal (Wehr et al., 2001) may be of

importance. It also remains unclear as to why some persons are susceptible and not others. More recent theories suggest a combination of pathophysiologies with an emphasis on the ANS and energy conserving behaviour.

The Hibernation Argument

SAD has been compared to hibernation because of the similarities in symptomology (Rosenthal et al., 1984; Mrosovsky, 1989). Symptom similarities between SAD and hibernation include hypersomnia, lack of energy, change in food preference, overeating, and weight gain. Many individual SAD sufferers liken their winter experience to hibernation with comments including “feeling like a hibernating bear”; “entering a little hibernation”; “I should have been a bear. Bears are allowed to hibernate; humans are not.”; “. . . prepare for winter . . . like a squirrel about to hibernate” (Rosenthal, 1993). Further, in a single case study, Harrison (1997) describes an individual who felt she was in “hibernation” and experienced the atypical behavioural symptoms during winter without depression. In spring, when the symptoms lifted, the individual felt “zippy”.

Several differences between SAD and hibernation include changes to sleep and temperature. Large decreases in temperature and heart rate are evident in mammals, though any decrease in SAD sufferers is minimal (Krueger & Shoham, 1986). The relationship between food intake, weight gain, and inactivity also differs. Mammals respond spontaneously to changes in the environment. Many species prepare for winter, with nest building, food hoarding, and weight gain preceding hibernation (Krueger & Shoham). On the other hand, social influences

are more important for humans (Kleitman, 1949), with eating and weight gain *throughout* winter. SAD sufferers are required to adapt their daily schedules of sleep, work, meals, and leisure with changes to the 24-hour cycle of light and darkness. During winter, with shorter daylight hours, SAD sufferers increase their daily hours of sleep.

Physiological similarities between SAD and hibernation include: an onset related to the variation in the daily photoperiod (Kleitman, 1949; Wehr et al., 2001). In mammals, entry into hibernation is spontaneous in response to a photoperiodic cue (Nicol & Andersen, 1996). Until recently a biological cue for seasonal change in humans was unknown. However, a recent finding is that SAD patients may generate a biological signal that registers the change in photoperiod similar to that in animals (Wehr et al.). Whilst the hibernation process differs amongst species of mammals, the purpose is to adapt to the colder months as an energy saving mechanism (Malan, 1996). Similarly, SAD has been described as energy conserving (Putilov & Danilenko, 1998).

Studies involving the cardiovascular system and the role of SNS and PNS in hibernation have been documented. Parasympathetic activity slows the heart for entry into hibernation (Burlington & Milsom, 1989; Lyman, 1982), then has minimal or no involvement during deep hibernation. The SNS is involved during hibernation maintaining cardio-vascular tone, and also in the process of arousal from hibernation (Lyman & O'Brien, 1963). The SNS also maintains vasoconstriction during hibernation (Burlington & Milsom, 1989).

Recent advances in SAD implicate metabolism / energy conservation (chapter 4), vagal tone, and hypoarousal, as being important factors in its pathophysiology, and also support the hibernation/ SAD comparison. SAD patients showed lower oxygen consumption, and lower resting metabolic rates during their depressed phase as compared to controls (Putilov, 1998). Further, Putilov & Danilenko (1998) suggest that the therapeutic action of light therapy is its energising effect that activates the sympatho-adrenal system, a finding consistent with arousal from hibernation.

Some researchers, for example Mrovosky (1989) acknowledge the similarities between hibernation and SAD, but feel the comparison is unhelpful and unnecessary. Because the hibernation process differs among animals, Mrovosky warns of the danger of choosing the most appropriate model and applying it to SAD implying similar mechanisms. On the other hand, the finding that the energy conserving nature of SAD may be of critical importance suggests that the hibernation/ SAD comparison may be a crucial one in further understanding a pathogenesis and underlying mechanisms in SAD, and may assist in therapy options.

In summary, whilst early similarities in symptomology between SAD and hibernation were noted, caution was also expressed by some researchers who indicated that the similarities did not mean there were similar underlying mechanisms. More recent advances in understanding both the hibernation process,

and pathophysiologies underlying SAD, suggest further investigation to be warranted.

Conclusions

Light therapy is successful in reversing the symptoms of SAD, and approaches to determine the mechanism of action involved may also assist in understanding mechanisms in SAD. Several theories have been proposed of the basis of findings from experimental light therapy in SAD, with the most recent research suggesting a combination of pathophysiologies is most likely. For example, evidence for circadian rhythm disturbances, biochemical abnormalities in neural pathways, and changes to autonomic nervous system functioning have been shown to contribute to the pathophysiology in SAD. Increased vagal tone during winter was shown in S-SAD (Austen & Wilson, 2001) contrasting with the decreased vagal tone typical of NSD (e.g., Rechlin et al., 1994), offering support for a further distinction between the two depressive disorders. Any suggestion of comparison with hibernation is tentative, however several similarities (e.g., increased need for sleep, weight gain, and low energy levels) warrant further investigation.

Part 11

The Experimental Phase

Chapter 7

Longitudinal Study 1 - Autonomic arousal in SAD

Chapter 7: Longitudinal Study 1 - Autonomic Arousal in SAD

The atypical vegetative symptoms of SAD (e.g., hypersomnia, increased appetite, weight gain, lack of energy) represent low levels of arousal that may be an attempt to conserve energy through the autumn/ winter months. The symptoms also appear consistent with increased parasympathetic or decreased sympathetic nervous system activity. A pilot study (Austen & Wilson, 2001) conducted as a forerunner to the current longitudinal research showed vagal tone to be high during winter in S-SAD giving initial support for the proposition. The finding of increased vagal tone was suggested to represent the sleep symptom rather than negative mood, and as such may show similarities with the hibernation process. Chapter 4 of this thesis showed the metabolic symptoms, energy, weight, and sleep, to contribute to 87% of the shared variance in the global seasonality score, and also suggests similarities with the hibernation process in animals, warranting further investigation. Whilst mechanisms of hibernation differ amongst mammals, parasympathetic activity has been shown to be high during entry into the hibernation state (Burlington & Milson, 1989). Sympathetic arousal is low maintaining cardiovascular tone during hibernation. Sympathetic activation enables arousal from hibernation in spring.

Respiratory sinus arrhythmia (RSA), is the rhythmic fluctuation in HR at each respiration cycle; HR accelerates with inspiration and decelerates with expiration (Bernston, Cacioppo, & Quigley, 1993). RSA is sensitive to behavioural and cognitive changes. For example, RSA is high during sleep (Porges, 1995), enhanced with aerobic fitness (de Geus, van Doornen, de Visser, & Orbeleke,

1990), and reduced under conditions of stressful task performance (Porges). RSA is also reduced with negative emotion (Porges). For example, RSA is withdrawn in NSD (Balogh et al., 1993; Rechlin et al., 1994; Rechlin et al., 1995), a finding that is *opposite* to the increased vagal tone shown in subsyndromal SAD by Austen and Wilson (2001).

RSA was originally thought to index a global measure of cardiac vagal tone, and to provide a non-invasive index of parasympathetic nervous system activity (Bernston et al., 1993; Lacey, 1976). Inconsistencies in the relationship between RSA and HR suggest the assumption of a single vagal source may be incorrect. A more recent theory, Porges' (1995) Polyvagal Theory, suggests that vagal efferent fibres originate primarily in two medullary nuclei, the nucleus ambiguus (NA) and the dorsal motor nucleus (DMNX). These two vagal mechanisms affect RSA and HR respectively. The two vagal systems have different response strategies that may operate independently of each other. Early research (e.g., Bernston et al., 1993; Lacey, 1976) suggested a reciprocal control of the two branches of the autonomic nervous system. It was believed that where one branch was decreased, then the other was increased. The literature now indicates that parasympathetic and sympathetic activity may co-vary independently of each other (e.g., Bernston, Cacioppo, Quigley, & Fabro, 1994), suggesting the need to measure indexes of both parasympathetic and sympathetic activity. Whilst RSA gives a pure measure of parasympathetic activity, measures of sympathetic activity are not so well defined. For example, HR is dually innervated by both sympathetic and parasympathetic branch of the autonomic nervous system.

A large body of research has been conducted to determine a pathogenesis in SAD. However, whilst a clearer picture is emerging with recent theories (e.g., Putilov & Danilenko, 1998) suggesting a combination of pathophysiologies is involved, a pathogenesis is still unknown. It is also unclear why some individuals are vulnerable to developing SAD and not others. An initial pilot study (Austen & Wilson, 2001) has shown initial support for the proposition that the atypical vegetative symptoms of SAD represent low levels of arousal. This finding suggests that SAD may have differing underlying mechanisms from NSD and provides a further similarity with hibernation. Further, results from Chapter 4 of the thesis show the importance of the metabolic symptoms, warranting further investigation of autonomic activity in SAD. Thus the aim of the current longitudinal study is to determine autonomic activity in SAD at clinical and subsyndromal levels, and control participants at bimonthly intervals across a complete annual cycle. Additional autonomic and constitutional measures were taken to support the proposition of hypo-arousal in SAD as well as to support previous findings. The measures taken were; heart rate (HR), skin conductance level (SCL), pulse height, blood pressure, sublingual temperature, skin temperature, percentage body fat, and body mass index. Measures were taken during baseline and also during a mental arithmetic stressor task. Aerobic fitness was measured at each bimonthly testing session using a bicycle ergometer test to control for any differential effect of fitness on RSA and HR between the groups (de Geus et al., 1990).

The experience of arousal has also been determined by using the Tension and Effort Stress Inventory (TESI) (Svebak, 1993) that measures eight pleasant or positive emotions and eight unpleasant or negative emotions identified in the

Reversal Theory (Apter, 1993). Of these, four positive and four negative emotions are somatic/ body emotions moderated by felt arousal. The remaining four positive and four negative are transactional emotions moderated by the outcome of transactions with others. The reasons for administration of the trait version of the TESI were twofold. Firstly, to determine the levels of tension stress felt from four sources, as well as effort stress to enable coping. Secondly, the measurement of the range of sixteen Reversal Theory emotions will determine experiences from arousal and from transactional outcomes. An additional scale from the state version of the TESI, current felt stress, enabled affirmation of the mental arithmetic task as a stressor for the psychophysiological variables. The Motivational State Profile (MSP) measures the strength of five pairs of motivational states, and two pairs of motivational tendencies.

Depressive mood and atypical vegetative seasonal symptoms were predicted to be present for both SAD groups during the months of May and July representing their depressed mood and associated behavioural symptoms during the autumn and winter months, with greater depression for C-SADs than for S-SADs. For all remaining months, the SAD groups were not expected to differ from the control group.

RSA was predicted to increase and HR to decrease for the SAD groups in winter supporting the proposition of increased parasympathetic activity. Additional sympathetic measures were predicted to decrease for winter representing low arousal. Higher body mass index and percentage body fat for May and July were predicted reflecting the weight gain symptom.

RSA was predicted to decrease, and HR and SCL to increase with cognitive stressor conditions for all participants irrespective of month or group. Winter maximums were expected in SCL, blood pressure, to confirm previous findings.

The TESI and MSP have not previously been used either in SAD, or in control participants across a twelve-month period. However, the SAD groups may be expected to experience higher levels of the negative emotions and lower levels of the positive emotions during the autumn/ winter months, as a reflection of their depressed mood.

Method

Participants

Psychology 1 students at the University of Tasmania completed the Seasonal Pattern Assessment Questionnaire (SPAQ) (Rosenthal et al., 1984) in early autumn of 1998 and 1999. The SPAQ screens seasonal change in mood and behaviour (Appendix A) and is more fully described in Chapter 4. The sum of six scales (sleep length, social activity, mood, weight, appetite, and energy level) produces a Global Seasonal Score (GSS). S-SAD participants were selected on the basis of a moderate to high GSS, together with their reported peak of symptoms in the winter months. S-SAD participants also satisfied Rosenthal's (1984) criteria for the SAD symptoms with depressive episodes that did not meet criteria for a Major Depressive Episode (American Psychiatric Association, 1994). C-SAD participants were selected on the basis of a high GSS together with their reported peak of symptoms in the winter

months. C-SAD participants also satisfied Rosenthal's criteria for SAD upon informal interview prior to the first testing session. Control participants were selected on the basis of a low GSS, and not responding with changes in mood and behaviour across the seasons. Additional C-SAD and S-SAD participants satisfying the selection criteria were obtained by advertising within the University of Tasmania, the local newspaper, and local media.

Sixty-one participants completed all six bimonthly testing sessions. There were 22 control participants (GSS $M = 2.36$, $SD = 2.04$, range 0 – 6), 21 S-SAD participants (GSS $M = 11.38$, $SD = 2.16$, range 8 – 15), and 18 C-SAD participants (GSS $M = 16.72$, $SD = 2.61$, range 13-22). Mean ages were, for controls, $M = 22.18$, $SD = 8.50$, age range was 17 to 46; for S-SADs, $M = 27.29$, $SD = 10.37$, age range was 18 to 48; and for C-SADs, $M = 28.72$, $SD = 10.20$, age range was 18 to 47. The female: male ratio was 4:1 which is representative of the gender distribution in the Psychology 1 classes.

All participants were Tasmanian residents for a minimum of three years prior to completion of the screening questionnaire to control for climatic adjustment at latitude 43° South. Ethical approval for the research was obtained from the University Ethics Committee (Human Experimentation) prior to laboratory testing. Written informed consent obtained from participants (Appendix B).

Apparatus

Psychological measurement. The Beck Depression Inventory with Addendum (BDIadd) and the Weekly Mood Inventory (WMI) were administered to

enable validation of seasonal depression. A Sleep Quality and Food Preference Questionnaire (SQFP) was administered to determine participants' quality of sleep and any change in food preference. The Tension and Effort Stress Inventory (TESI) and the Apter Motivational Style Profile were administered to determine experience of stressors, related efforts to cope, the assessment of a range of pleasant as well as unpleasant moods, and metamotivational states.

The BDIadd is a self-rating scale for depression comprising of the Beck Depression Inventory (BDI) (Beck & Steer, 1987), and an addendum (Meesters & Jansen, 1993, personal communication, March 29, 1995) for assessing seasonal depression. The addendum included items 12 (social withdrawal) and 17 (fatigability) from the BDI as well as items 16 (hypersomnia), 18 (appetite), and 19 (weight gain) which were inversely formulated.

The WMI (Rosenthal et al., 1984) (Appendix E) is a self rated 7-point scale (0 = not at all, 6 = very much) developed for the assessment of SAD patients and requesting information about major life events, illness, and mood during the previous week. Factors, Depressed Mood and Ideation (DMI), Atypical Vegetative Symptoms (AVS), Typical Vegetative Symptoms (TVS), and Hypomania Mood and Ideation (HMI), were based on the clinical findings of others. The internal cohesiveness of the factors is high, ranging from 0.76 to 0.89 (Rosenthal et al., 1989).

The SQFP (Appendix F) comprises four 7-point scales developed in the School of Psychology at the University of Tasmania to determine sleep patterns in

SAD, and to determine any change in food preference at bimonthly intervals across a twelve month period. Participants recorded their sleep quality according to four 7-point scales (good nights/bad nights, restful sleep/disturbed sleep, slept lightly/slept deeply, and felt refreshed/felt fatigued). Participants also recorded whether they experienced any change in food preference (yes/ no) over the previous month. Where a 'yes' response was recorded, participants recorded the change in food preference from four choices (fats, protein, carbohydrates, other). More than one choice was permitted.

Constitutional measures. Height (m) and weight (kg) were measured using a Kawe (Germany) wall-mounted height measure and Tanita electronic digital scales, respectively. Blood pressure was measured using a Sein portable Digital Blood Pressure Meter (Model SE-2000) and sublingual temperature taken using a Sharp Digital thermometer (Model MT 20). Skinfold thickness was measured at four sites; biceps, triceps, subscapular, and suprailiac, using John Bull British Indicators Ltd. Skinfold Callipers according to Durnin and Womersley (1974). Submaximal fitness level was assessed by a six-minute period of exercise on a Repco bicycle ergometer. Oxygen (O₂) uptake was calculated using the nomogram of Åstrand (1960).

Psychophysiological measurement. Psychophysiological recordings used a MacLab Data Acquisition System with Chart 3.4 software on a PowerMac 7300 computer. The system was configured with six channels to record electrocardiogram (ECG), cardiometer, finger pulse amplitude, respiration, skin conductance level, and skin temperature. ECG standard Ag/ AgCl electrodes were

filled with electrode gel and attached with adhesive collars to L-rib, R-rib placements and mastoid earth. A Bioamplifier couple was used for ECG with a band-pass filter of 0.3 Hz to 50 Hz. Cardiometer readings were computed directly from the ECG channel. A photo-electric finger pick-up, attached to the distal phalange of participant's second finger was used to measure finger pulse amplitude, and processed by a General Purpose coupler with a band-pass filter of 0.3 Hz to 10 Hz. A Vitalog Respiratory Chest Transducer belt, and Vitalog Respiration and Body Position Amplifier, were used for respiration recording, and connected directly to the MacLab channel input. The respiratory pacing used a four-channel interval generator delivering 2 tones (1 kHz and 1.5 kHz) using Sonalerts. Each complete cycle was 6 secs (10 breaths per minute). The paced breathing used respiratory cycle times of 7.5, 6, 5, 4, and 3 secs for 8, 10, 12, 15, and 20 breaths per minute respectively. Eight mm Ag/AgCl electrodes for skin conductance were filled with electrode gel and attached with surgical tape to the second phalanx of the participant's first and second fingers (Venables & Christie, 1973) and connected to a GSR Amplifier with a range of 10 μ S applied. Two thermocouples and a Digitron Digital Thermometer (Model 2751-K) were used for skin temperature and connected directly to the MacLab Data Acquisition System with a low pass filter of 50 Hz. One thermocouple was attached to the distal phalanx of the first finger with surgical tape, and the other directly over the left superficial temporal artery about 1 cm above the zygomatic bone (Harrison, 1990). The participant's non-dominant hand was used for FPA, SCL, and finger skin temperature.

The six-channel configuration used different sampling rates for each channel. Sampling rates were 200 cycles per second for the ECG, cardiometer,

and FPA channels, 100 cycles per second for respiration, and 10 cycles per second for SCL and Skin Temperature.

Stressor task. The psychophysiological stressor was a Mental Arithmetic task (MA) with software installed on a PC computer. Twenty practice trials were given, then two test blocks of thirty trials each. In each trial an arithmetical problem using 2 double-digit numbers appeared on the screen. The participant was required to respond with the correct answer from a four-choice panel. Maximum response time allowed for each trial was 6000 ms. From the sixty test trials thirty-two problems involved addition, and twenty-eight involved subtraction.

Emotional measurement. The trait version of the TESI (Svebak, 1987) has three parts using 7-point scales to rate the experience of stressors and emotions in the thirty days prior to each testing session. For Part A, participants estimated the degree of stress/ or pressure (1 = no pressure, 7 = very much) they had been exposed to from four stress sources (work, family, finance, own body). For Part B, participants estimated the degree of effort (1 = no effort, 7 = very much) expended to cope with each of the four stress sources. Part C consisted of the assessment of the sixteen emotions proposed by the Reversal Theory (Apter, 1993). Participants estimated the degree (1 = not at all, 7 = very much) to which they had experienced each emotion over the previous thirty days. Of these, there were four emotions in each of four categories: positive somatic (relaxation, excitement, placidity, and provocativeness), negative somatic (anxiety, boredom, anger, and sullenness), positive transactional (pride, modesty, gratitude, and virtue), and negative transactional (humiliation, shame, resentment, and guilt).

An additional 7-point scale (1 = no stress, 7 = high stress) from the state version of the TESI was included. Participants rated the degree of stress they were feeling (a) at the commencement of each session and (b) mid-mental arithmetic task.

The Apter Motivational Style Profile (MSP) (Apter, Mallows, & Williams, 1998) measures dominance and is a 70-item self-rated 7-point scale which measures dominance and salience in five pairs of metamotivational states identified in the Reversal Theory (Apter, 1993) as well as motivational tendencies towards arousability/ effortfulness and optimism/ pessimism.

Procedure

Participants were tested individually. The laboratory was sound attenuated and temperature controlled (24°C). Half of the participants were tested in the morning (8.00 a.m. to 12 noon) and half in the afternoon (2.00 p.m. to 6.00 p.m.) to control for any circadian differences in physiological response levels and performance measures. Counterbalancing was across group and gender. Participants were requested to refrain from eating, and caffeine or nicotine ingestion for one hour prior to testing.

Each participant was tested on six occasions at bimonthly intervals. Testing sessions were in January, March, May, July, September, and November. All experimentation was completed over a three-year period. Consent form (Appendix B) and health checklist (Appendix D) were completed at the first session.

Tasmania has daylight savings where clocks are put forward one hour from first Sunday in October to the last Sunday in March (Tourism Tasmania, (On-line), 2001). Participants were not tested in the first two-week period after the commencement, or, after the end, of the daylight savings period to allow for adjustment of participants' circadian rhythms.

Each laboratory session commenced with height, weight, and skinfold thickness measures. HR and SCL electrodes were attached and the respiration band fitted around participants' upper chest while inspiring. The participant was then seated at a PC computer with photoelectric finger pick-up and skin temperature thermocouples were attached. In addition, sublingual temperature and blood pressure were measured. Finger and face skin temperature used digitron thermocouples manual switching by the experimenter at the beginning of each session, and again during the stressor task. Responses were recorded on the MacLab Data Acquisition System in an adjoining room where the experimenter monitored psychophysiological responses. The participant then completed four questionnaires, BDIadd, WMI, SQFP, and the TESI. On completion of the questionnaires, breathing was paced at ten breaths per minute for a period of at least one minute while baseline psychophysiological measures were recorded, then the two blocks of the Mental Arithmetic stressor task were given. Standard instructions were given for the Mental Arithmetic task, and practice trials completed. At the end of Mental Arithmetic Block 1, the 7-point scale for current felt stress from the TESI was repeated to determine the stress load created by the stressor task, and breathing paced at ten breaths per minute for a further period of one minute. On completion of the stressor task paced breathing was given. Breathing was paced for approximately one minute at eight, ten, twelve, fifteen, and twenty breaths per minute. The

respiration band, finger electrodes, photo-electric pick-up, and thermocouplers were removed. For the bicycle ergometer fitness test HR was monitored. The workload on the bicycle ergometer was set according to the participant's normal level of physical activity and the test was terminated after six minutes.

The Apter Motivational Style Profile was administered twice: at the end of the testing session in January (summer) and in July (winter).

Design

Laboratory testing commenced with twelve participants (four control, four S-SAD, and four C-SAD). Four additional participants were added to each group at each consecutive bimonthly laboratory session, with each participant completing six testing sessions. Due to the longitudinal nature of the experiment, many participants were unable to complete their six bimonthly sessions. Replacement participants were added at the next opportunity. The difficulty of retaining SAD participants, particularly through their 'at risk' autumn/ winter period was greater than that of control participants.

All dependent variables used a mixed between and within subjects design. The between groups factor was Group (Control, S-SAD, and C-SAD). The within groups factor for all dependent variables was Month (January, March, May, July, September, and November). The psychophysiological RSA, HR, SCL, and skin temperature measures, as well as the TESI current felt stress scale also used Condition (Baseline, Stressor task) as a within groups factor.

Data scoring and analyses

Trend analyses using planned polynomial contrasts were conducted for Month, and Month x Group, on each dependent variable using SPSS 10 specifically to estimate any quadratic and/ or cubic seasonality trend across the twelve-month period (Maxwell & Delaney, 1990). A quadratic curve for month represents either a peak or a trough during the twelve-month period. A significant cubic curve represents a secondary peak or trough as well. Where polynomial contrasts revealed a significant quadratic or cubic interaction for Month x Group, polynomial contrasts were conducted for each group individually to locate the significant seasonality effect.

Omnibus multivariate repeated measures analyses were also performed on all dependent variables, except for the TESI analyses. MANOVAs were used following Vasey and Thayer (1987) as the most appropriate analyses for repeated measures where an adequate sample size for the number of repeated measure is available. Pillai's criterion was chosen as the multivariate test as it is more robust with unequal groups, and the assumption of homogeneity of variances violated (Tabachnik & Fidell, 1989). Where multivariate analyses revealed a significant main effect or interaction, follow up with univariate analyses and LSD post hoc pairwise comparisons were performed where appropriate using two-tailed t-tests to identify specific significant effects revealed in the omnibus MANOVAS or ANOVAS. Analyses used SPSS 10 for Windows. The significance level used was $\alpha = .05$.

Because of the large number of repeated measures in the TESI relative to numbers of participants in groups, ANOVAs were used for all TESI analyses and epsilon adjustment for repeated measures was made using the Greenhouse Geiser correction procedure (Vasey & Thayer, 1987).

Screening questionnaire. One-way between groups ANOVAs were conducted on GSS, GR, seasonal weight fluctuation, and age. Multivariate analyses were conducted for the SAD symptoms and sleep. A one-way between groups MANOVA was conducted on the degree of change across seasons for the six symptoms: appetite, energy level, mood, sleep, socialisation, and weight; as a relationship between the symptoms was not required (Tabachnik & Fidell, 1989). A 3 x 4 (Group x Season) repeated measures MANOVA was conducted on the mean number of hours of sleep recorded in a 24-hour period for each season. The within subjects factor for hours of sleep was Season (spring, summer, autumn, and winter). The between groups factor for all screening questionnaire variables was Group (Control, S-SAD, and C-SAD). Post hoc pairwise comparisons were performed where necessary to identify the source of significant main effects and interactions.

Depression and behavioural symptoms. The BDIadd, the four WMI subscales, and the four sleep quality subscales from the SQFP, each used a 6 x 3 (Month x Group) repeated measures MANOVA. For Food Preference, the percentage of each group reporting changes to food preference was calculated for each of the four choices (fats, protein, carbohydrates, and other). No additional statistical analyses were performed on the food preference measures.

Constitutional measures. BMI was calculated using the formula: mass (kg)/height² (m). Percentage body-fat was calculated as the sum of four skin-fold thicknesses (biceps, triceps, sub-scapula, and suprailiac), then converted to a ratio of body weight according to age and sex (Durnin & Womersley, 1974). A measure of O₂ uptake was calculated from average HR the last 15 seconds of the 6-minute sub-maximal fitness test using to the nomogram of Åstrand (1960).

Multivariate repeated measures analyses were used for all constitutional measures. BMI, systolic and diastolic blood pressure, sublingual temperature, percentage body-fat, and O₂ uptake were analysed using a 6 x 3 (Month x Group) repeated measures MANOVA. LSD post hoc pairwise comparisons were used to locate significant differences when there were significant main effects and interactions.

Polynomial contrasts were also used for each constitutional variable. Where polynomial contrasts revealed a significant quadratic interaction between Month and Group, further polynomial contrasts were conducted on each group individually to locate the significant seasonality effect.

Psychophysiological measures. Actual RSA was calculated using the peak-trough method (Porges & Byrne, 1992): actual RSA was the difference between maximum and minimum HR associated with each respiratory cycle averaged over five consecutive breaths. Predicted RSA was calculated by substituting the respiration rate of 10 breaths per minute for the baseline condition into a linear regression function for each participant from their paced breathing. For the stressor condition respiration rates from each individual recording were

substituted into the individual regression equation for the paced breathing calibration levels. Predicted RSA was subtracted from actual RSA to produce a difference score. This RSA difference score reflects the cognitive load component in processing information independent of possible respiratory changes during the experimental procedure. HR, SCL, and skin temperature were taken from the same five-breath period as RSA. Pulse height was derived from the FPA channel, and a score calculated as a % of baseline: pulse amplitude scores of over 100% indicated vasodilation has occurred.

Multivariate repeated measures analyses were used for all psychophysiological measures. HR, RSA, SCL, and finger and face skin temperature were analysed using a $6 \times 2 \times 3$ (Month \times Condition: baseline/ stressor \times Group) repeated measures MANOVA. Pulse height was analysed using a 6×3 (Month \times Group) repeated measures MANOVA.

Stressor task. Response time scores were calculated for addition and subtraction trials separately. The two blocks of mental arithmetic were averaged. Multivariate repeated measures analyses were used for the Mental Arithmetic stressor task. Response time scores for both addition and subtraction were analysed separately using 6×3 (Month \times Group) repeated measures MANOVA.

Tension and Effort Stress Inventory. There were 4 types each of tension stress and effort stress (work, family, finance, somatic), which were analysed using a $6 \times 2 \times 4 \times 3$ (Month \times Stress type: tension stress/effort stress \times Stress Source: work, family, finance, somatic \times Group) repeated measures MANOVA. Ratings for 16 emotions were compiled into four categories: positive somatic emotions

(excitation, relaxation, placidity, and provocativeness); negative somatic emotions (boredom, anxiety, anger, and sullenness); positive transactional emotions (virtue, pride, gratitude, and modesty); and negative transactional emotions (humiliation, shame, resentment, and guilt). The emotions were analysed using a $6 \times 4 \times 3$ (Month \times Emotion \times Group) repeated measures MANOVA. Main effects for emotions and stressors from the TESI are meaningless and will not be described; however, interactions involving emotions and stressors are important and interpretation will be made. Current stress felt was rated twice: at the commencement of each session, and after the stressor task. Current stress was analysed using a $6 \times 2 \times 3$ (Month \times Time: pre/post stressor task \times Group) repeated measures MANOVA.

The Motivational Style Profile. Items were scored using standard procedures to produce subscales for each motivational style pair (telic/ paratelic; arousal-avoiding/arousal-seeking; defiant/compliant; autic mastery/ autic sympathy; alloic mastery/ alloic sympathy) and motivational tendency (optimism/pessimism, and emotionality/ effortfulness) according to Apter, Mallows, and Williams, 1998. Multivariate $2 \times 2 \times 3$ (Season \times Motivational state \times Group) repeated measures analyses were used for each pair of motivational states. Multivariate $2 \times 2 \times 2 \times 3$ (Season: summer/winter \times Mastery: mastery/sympathy \times Autic: autic/alloic \times Group) repeated measures analyses were used for the four transactional variables. Polynomial contrasts were not conducted on the data, as the MSP was administered twice only.

Results

Screening questionnaire

Means and standard deviations for self-rated scores from the Seasonal Pattern Assessment Questionnaire are presented in Table 8.

Univariate analyses. One way between groups ANOVAs were performed for Global Seasonality Score (GSS), Global Rating (GR), Weight Fluctuation (WtFluct), and age. Main effects were revealed for GSS, GR, and WtFluct.

The main effect for GSS, $F(2, 58) = 209.03, p < .001$, indicates highly significant differences between the groups. Post hoc analyses showed highly significant differences between all pairwise group comparisons at the $p = .001$ significance level. Table 8 shows C-SADs and S-SADs to have a significantly higher GSS than controls. C-SADs also had a significantly higher GSS than S-SADs.

The main effect for GR, $F(2, 58) = 26.32, p < .001$, indicates highly significant differences between the groups. Post hoc analyses showed highly significant differences between all pairwise group comparisons at the $p = .001$ significance level. C-SADs and S-SADs had a significantly higher GR than controls. The GR for C-SADs was also significantly higher than for S-SADs.

Table 8

Means and Standard Deviations for Scores on the Screening Questionnaire (Seasonal Pattern Assessment Questionnaire) for Control, Subsyndromal SAD, and Clinical SAD groups in Longitudinal Study 1: Autonomic Arousal

	Source	Control		Subsyndromal SAD		Clinical SAD	
		M	SD	M	SD	M	SD
Degree of seasonal change to:	Appetite	0.23	0.43	1.52	0.81	2.72	0.83
	Energy	0.41	0.50	2.33	0.80	3.33	0.49
	Mood	0.41	0.59	2.29	0.85	3.11	0.76
	Sleep	0.64	0.58	2.10	0.62	2.44	0.78
	Social	0.41	0.59	1.90	0.77	2.78	0.88
	Weight	0.27	0.55	1.24	0.62	2.33	0.84
Global Seasonality Score		2.36	2.04	11.38	2.16	16.72	2.61
Global Rating		0.05	0.21	0.67	1.24	2.33	1.28
Weight Fluctuation		1.45	1.18	2.24	1.56	2.72	1.30
Sleep Length	Autumn	8.20	1.05	8.29	1.49	8.83	1.50
	Winter	8.27	1.12	9.24	1.76	10.17	1.92
	Spring	8.09	1.07	8.05	1.32	8.17	1.34
	Summer	7.80	1.33	6.81	1.40	7.39	1.38
Age		22.18	8.5	27.29	10.37	28.72	10.20

Note. The degree of change in a twelve month period irrespective of season for each symptom ranged from from 0 (no change) to 4 (extremely marked change). Global Seasonality Score was the sum of the six symptoms. Global Rating was from 0 (not a problem) to 5 (disabling). Weight Fluctuation in a twelve month period was recorded on a scale from 1 to 6 where 1 = 0-2 kg, 2 = 2-3 kg, 3 = 4-5 kg, 5 = 8-10 kg, 6 = over 10 kg. Sleep length was the approximate number of hours of sleep per 24 hour period during each season. All participants completed the SPAQ during the early autumn.

The main effect for WtFluct, $F(2, 58) = 5.65$, $p = .006$, indicated significant differences between the groups. Post hoc analyses showed that the weight of C-SADs and S-SADs fluctuated significantly more than controls at the $p = .001$ significance level. The two SAD groups did not differ from each other in WtFluct. The one-way ANOVA for age was non-significant.

Degree of change across seasons. A one-way between groups MANOVA was performed on six dependent variables measuring the degree of change across the seasons to the six symptoms: appetite, energy, mood, sleep, socialisation, and weight. The MANOVA was highly significant, Pillai's Trace = 1.022, $F(12, 108) = 9.41$, $p = .001$. Subsequent univariate F tests on each dependent variable showed all symptoms to contribute to significant differences between the groups. Univariate F values were: appetite, $F(2, 60) = 62.66$, $p < .001$, energy, $F(2, 60) = 118.39$, $p < .001$, mood, $F(2, 60) = 72.35$, $p < .001$, sleep, $F(2, 60) = 43.59$, $p < .001$, social activity, $F(2, 60) = 52.25$, $p < .001$, and weight, $F(2, 60) = 46.60$, $p < .001$. Post hoc analyses showed significant differences between all pairwise group comparisons for all symptoms at the $\alpha = .001$ significance level, except sleep. The SAD groups showed a greater degree of seasonal change for each symptom than controls. C-SADs also showed a greater degree of change than S-SADs. For sleep, both SAD groups rated sleep change significantly higher than controls at the $\alpha = .001$ significance level but did not differ from each other. Figure 5 shows these differences between the groups for each symptom.

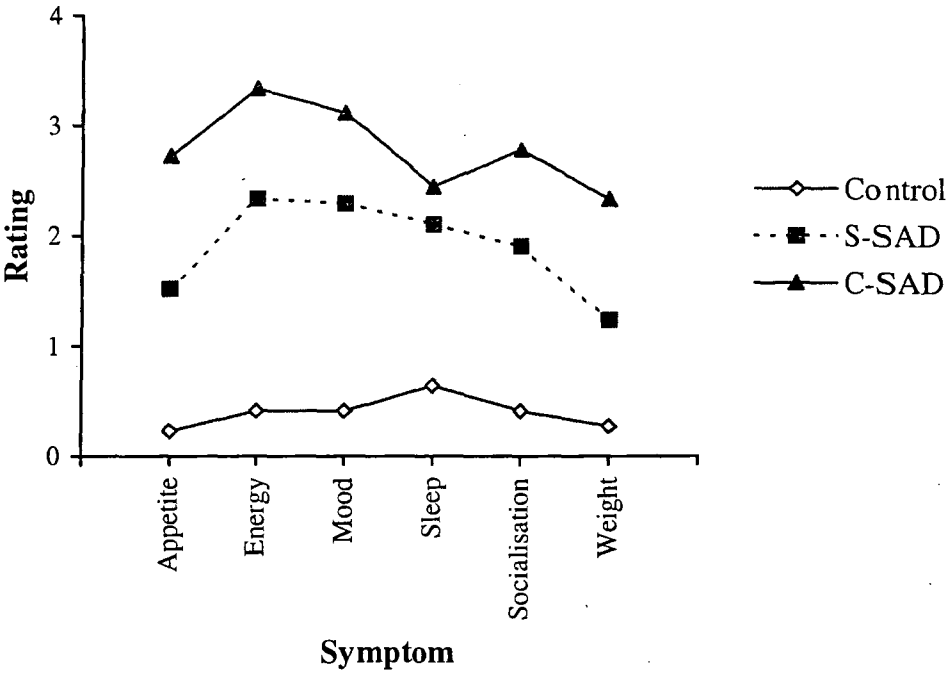


Figure 5. Degree of seasonal change for six SAD symptoms for control, subsyndromal SAD (S-SAD), and clinical SAD (C-SAD) groups on the Seasonal Pattern Assessment Questionnaire

Hours of sleep per twenty-four hour period. A 3 x 4 (Group x Season) repeated measures MANOVA was conducted revealing a main effect for Season, and a significant Group x Season interaction. The significant main effect for season, Pillai's Trace = .564, $F(3, 56) = 24.16, p < .001$, indicated significant differences between the seasons. The significant Group x Season interaction, Pillai's Trace = .426, $F(6, 114) = 5.14, p < .001$, indicated the groups differed in hours of sleep between the seasons. Post hoc pairwise comparisons show significant differences between the groups for winter only. C-SADs slept more hours than controls. S-SADs also showed a strong tendency to sleep more hours than controls ($p = .054$). Univariate repeated measures analyses were also conducted across the four seasons

for each group separately. Significant differences were revealed for C-SADs, and S-SADs, only. C-SADs slept more in winter than in spring, summer, or autumn, and also slept more in autumn than in summer. S-SADs also slept more in winter than in spring, summer, or autumn, and more in autumn than in summer. Controls did not differ in their hours slept between the seasons.

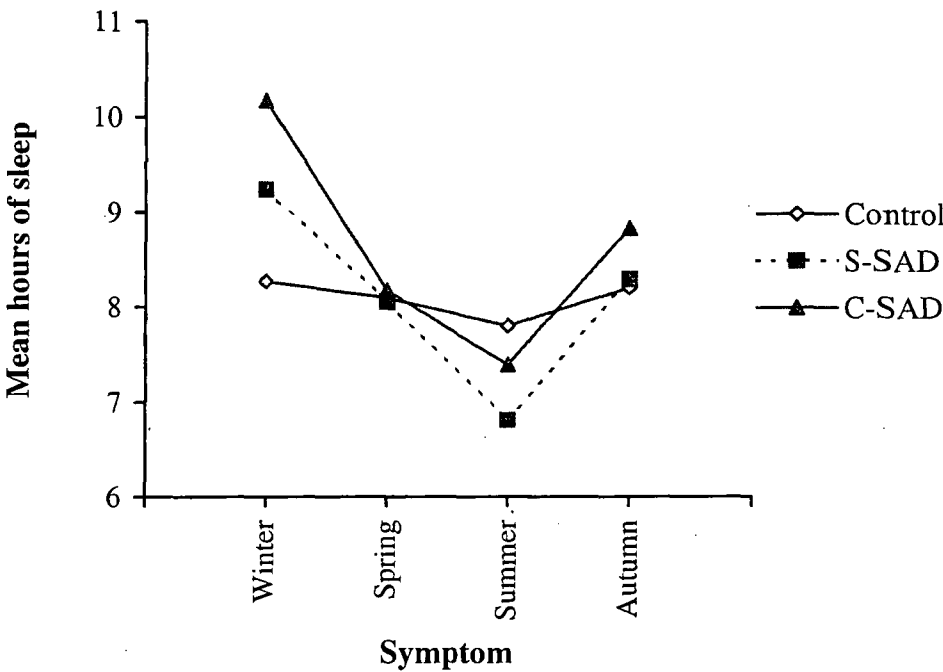


Figure 6 . Mean hours of sleep for control, subsyndromal SAD (S-SAD), and clinical SAD (C-SAD) groups for each season as reported on the Seasonal Pattern Assessment Questionnaire.

Summary of SPAQ results. The SPAQ screening questionnaire differentiates between the groups for GSS, GR, WtFluct, degree of change to each symptom: appetite, energy, mood, sleep, social, and weight, and hours slept in winter only. C-SADs also rated higher than S-SADs for all variables except WtFluct and degree of

change to the symptom, sleep change. The SAD groups also slept more in winter as compared to spring, summer, and autumn.

Depression and behavioural symptoms

Mean self-rated depression and behavioural symptom scores and standard deviations for BDIadd and WMI are presented in Table 9.

Beck Depression Inventory with Addendum. For BDIadd, a trend analysis using planned polynomial contrasts revealed a significant quadratic effect for month, and a significant cubic interaction between group and month.

The significant quadratic effect for month, $F(1, 58) = 22.64, p < .001$, indicated seasonality for participants overall, regardless of group. The significant cubic interaction between month and group, $F(2, 58) = 9.49, p < .001$, indicated that the seasonal pattern across the months varied between the groups. Subsequent polynomial contrasts conducted for each group separately revealed significant quadratic and cubic main effects for C-SADs, and a significant quadratic main effect for S-SADs. Figure 7 reflects this seasonal effect showing depression to peak in the autumn and winter groups for the SAD groups only. The C-SAD group also shows a small secondary peak for November.

A 6 x 3 (Month x Group) MANOVA was also conducted revealing main effects for month, and for group, and a significant interaction between month and group. The main effect for month, Pillai's Trace = .342, $F(5, 54) = 5.62, p < .001$, indicated a significant difference across the months.

Table 9

Means and Standard Deviations for Depression (Beck Depression Inventory with Addendum and Weekly Mood Inventory) for Control, Subsyndromal SAD, and Clinical SAD groups at Bimonthly intervals

		January		March		May		July		September		November	
	Group	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Beck Depression Inventory with Addendum	Control	1.95	3.40	2.64	6.95	3.00	5.71	3.36	5.23	3.18	5.64	1.82	4.10
	S-SAD	5.90	5.61	6.81	6.98	10.76	10.37	9.57	8.09	8.57	7.30	4.00	4.52
	C-SAD	4.67	4.54	7.33	4.67	12.28	7.98	10.33	7.62	6.94	4.26	9.50	8.30
Weekly Mood Inventory													
Depressed Mood and Ideation (6 items)	Control	5.59	5.93	5.68	7.01	8.32	6.83	6.82	6.85	7.55	7.04	5.55	6.12
	S-SAD	9.43	8.00	10.52	8.47	13.81	8.00	12.19	9.65	10.71	7.05	8.95	5.79
	C-SAD	7.67	6.45	9.72	5.39	15.33	8.18	12.67	7.13	10.78	6.70	11.94	7.98
Typical Vegetative Symptoms (5 items)	Control	5.41	4.87	6.73	6.83	7.91	6.29	6.18	6.69	6.36	6.53	7.59	6.34
	S-SAD	9.33	6.94	11.62	7.05	11.33	7.16	9.81	7.81	10.00	6.61	9.90	6.74
	C-SAD	8.28	5.57	13.22	5.49	11.11	5.82	10.67	6.58	11.89	5.66	11.22	7.45
Atypical Vegetative Symptoms (5 items)	Control	10.14	3.80	8.18	4.75	9.82	4.12	8.27	3.56	9.36	5.19	9.36	3.20
	S-SAD	9.19	5.99	10.81	4.58	12.95	5.58	13.57	6.98	10.57	5.04	9.76	6.05
	C-SAD	9.39	4.72	9.44	5.16	16.28	6.32	14.72	5.13	11.94	5.71	12.44	5.50
Hypomania Mood and Ideation (10 items)	Control	37.95	9.97	36.50	9.60	33.77	11.36	34.59	10.31	34.36	11.88	37.23	8.39
	S-SAD	34.62	6.79	30.57	9.07	28.90	11.23	30.67	10.17	31.90	10.50	36.19	11.15
	C-SAD	34.89	9.94	29.28	7.04	23.33	9.71	26.17	10.98	32.06	9.70	31.61	9.17

Note. Weekly Mood Inventory ratings were made on a 7-point scale and totalled for each factor.

n = 22 Control, 21 Subsyndromal SAD, and 18 Clinical SAD participants

The main effect for group, $F(2, 58) = 9.28, p < .001$, indicated a significant difference between the groups. MANOVA also showed a significant interaction between month and group, Pillai's Trace = .41, $F(10, 110) = 2.84, p = .004$. Figure 7 shows control, S-SAD, and C-SAD groups to vary differentially across the months.

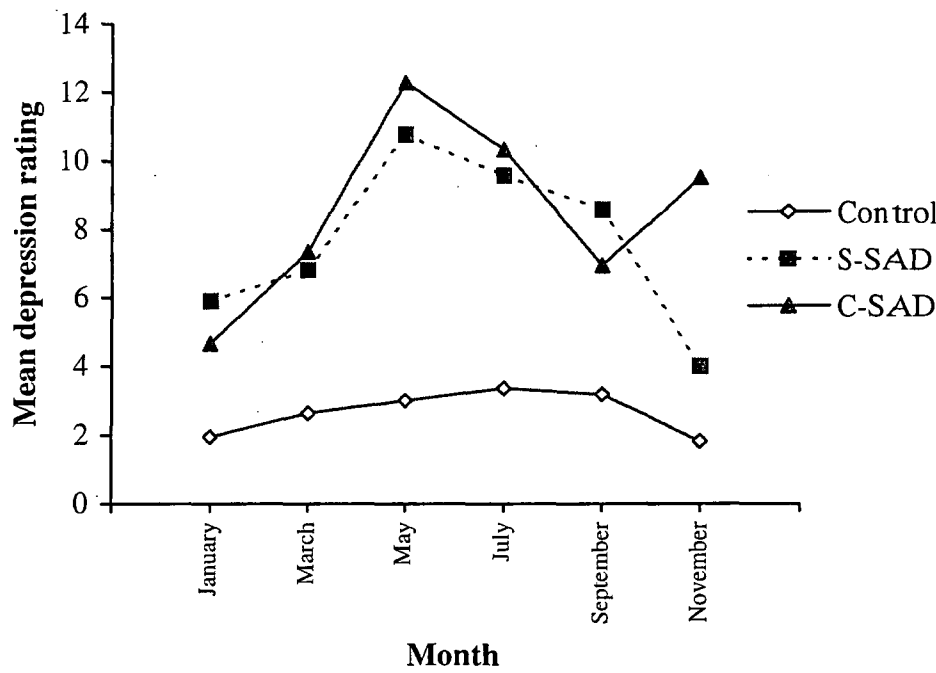


Figure 7. Depression ratings from Beck Depression Inventory with Addendum for control, subsyndromal SAD (S-SAD), and clinical SAD (C-SAD) groups at bimonthly intervals for a twelve-month period

The SAD groups rate depression significantly higher than controls in March, May, and July. C-SADs rate their depression higher in May than in January, March, and September. S-SADs rated depression significantly higher in May and July, than in January and November. Controls rated depression significantly higher in July and September, than in November.

Weekly Mood Inventory. For WMI, trend analyses using polynomial contrasts were performed separately for each subscale: depressed mood and ideation (DMI), typical vegetative symptoms (TVS), atypical vegetative symptoms (AVS), and hypomania mood and ideation (HMI). Overall, for DMI, trend analyses using polynomial contrasts revealed a significant quadratic main effect for month. For TVS, trend analyses revealed a significant cubic main effect. For AVS, trend analyses using polynomial contrasts revealed a significant quadratic main effect for month, and a significant quadratic interaction between month and group. For HMI trend analyses revealed a significant quadratic main effect for month.

Following the trend analyses, 6 x 3 (Month x Group) repeated measures MANOVAs were also conducted. Overall, for all WMI subscales, MANOVA revealed significant main effects for month, and for group. For AVS only, MANOVA also revealed a significant interaction between month and group.

For DMI, planned polynomial contrasts revealed a significant quadratic main effect, $F(1, 58) = 14.19, p < .001$, indicating a seasonal pattern across the months. Figure 8 shows DMI to peak for May. From the MANOVA for DMI, the main effect for month, Pillai's Trace = .292, $F(5, 54) = 4.46, p = .002$, indicated significant differences across the months. Univariate repeated measures analyses showed greater depressed mood for May than for all other months at the $p = .01$ level, and also greater depressed mood for July than January and March. The main effect for group, $F(2, 58) = 5.97, p = .004$, indicated significant differences between the groups. C-SADs and S-SADs had a significantly higher DMI than controls.

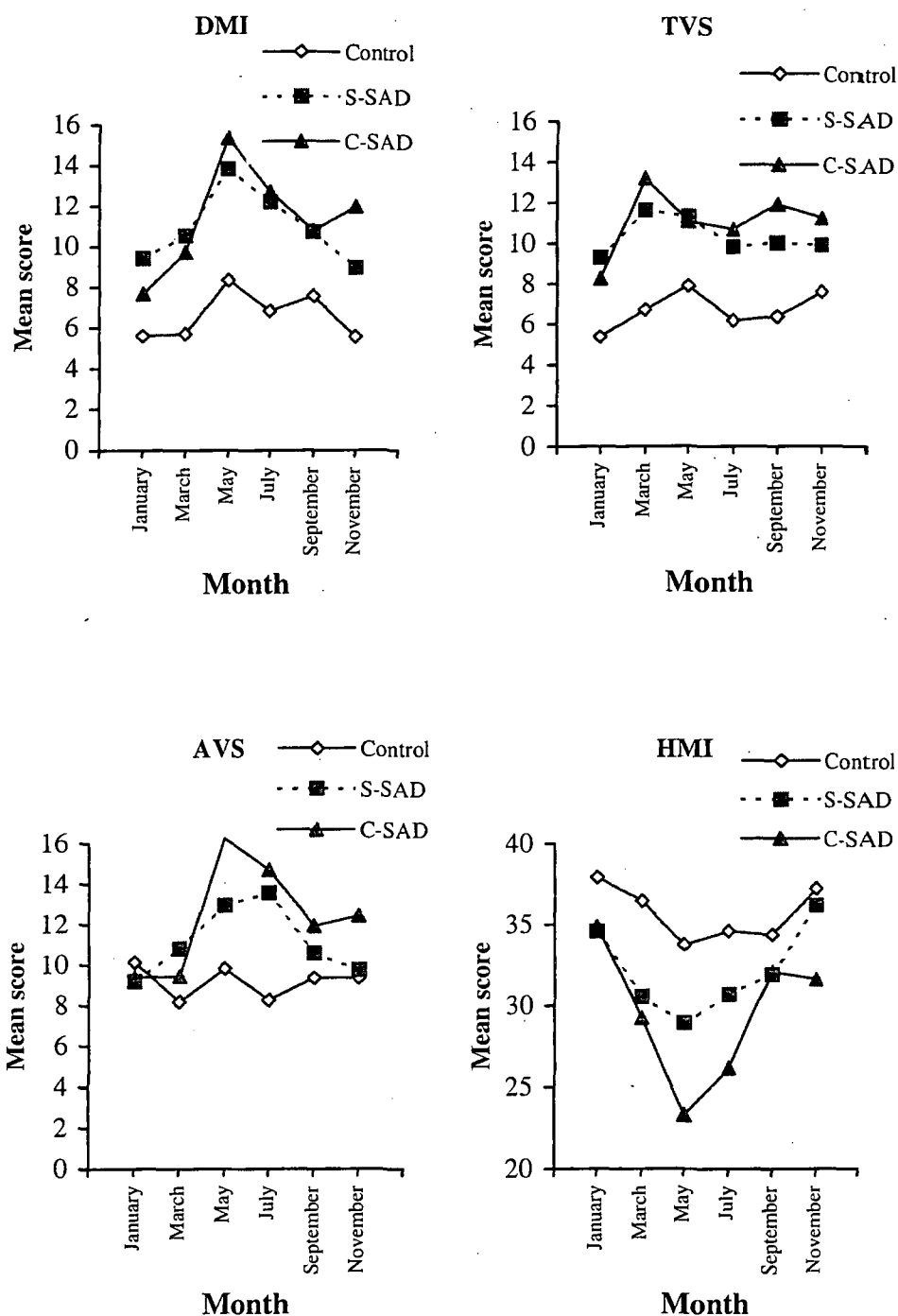


Figure 8. Weekly Mood Inventory Subscales: Depressed Mood and Ideation (DMI), Typical Vegetative Symptoms (TVS), Atypical Vegetative Symptoms (AVS), and Hypomania Mood and Ideation (HMI) for control, subsyndromal SAD (S-SAD), and clinical SAD (C-SAD) groups at bimonthly intervals across a twelve-month period.

For TVS, the significant cubic main effect, $F(1, 58) = 6.98, p = .011$, indicated a seasonal pattern across the months. Figure 8 shows a peak for TVS in March, and a secondary peak for November. From the MANOVA for TVS, the main effect for month, Pillai's Trace = .231, $F(5, 54) = 3.24, p = .013$, indicated the scores differed across the months. Typical symptom ratings were lower for January than for all other months except July. The scores were also lower for July than for March. The main effect for group, $F(2, 58) = 5.28, p = .008$, indicated the groups differed in non-seasonal typical depression symptoms. C-SADs and S-SADs scored greater depression than controls.

For AVS, planned polynomial contrasts revealed a significant quadratic main effect, $F(1, 58) = 11.61, p = .001$, indicating a seasonal pattern across the months. For AVS, planned polynomial contrasts also revealed a significant quadratic interaction, $F(2, 58) = 5.70, p = .006$, which indicated that the seasonal pattern varied between the groups. Subsequent polynomial contrasts for each group separately revealed significant quadratic effects for C-SADs and S-SADs. The interaction (Figure 8) shows the SAD groups to peak for May and July as compared to all other months.

The MANOVA for AVS revealed main effects for month, Pillai's Trace = .382, $F(5, 54) = 6.68, p < .001$, and for group, $F(2, 58) = 4.50, p = .014$. MANOVA also revealed an interaction between month and group, Pillai's Trace = .402, $F(10, 110) = 2.76, p = .004$, indicating that the groups differed in scores for seasonal symptoms across the months. Post hoc pairwise comparisons between the groups show significant differences for May and July. For May, C-SADs had

significantly greater seasonal symptoms than controls. In July, both SAD groups had greater seasonal symptoms than controls.

Subsequent univariate analyses across the months were conducted separately for each group revealing significant differences for C-SADs and S-SADs. C-SADs were significantly greater in May than in January, March, and September. C-SADs were also greater in July than in January and March. S-SADs were significantly greater in May than in January, and also greater in July than in January, March, and November. Figure 8 shows greater seasonal symptoms for the SAD groups in May.

For HMI, planned polynomial contrasts revealed a significant quadratic main effect, Pillai's Trace = .373, $F(1, 58) = 33.47$, $p < .001$, which indicated a seasonal pattern across the months. Figure 8 shows lower scores for HMI during the autumn and winter months. From the MANOVA for HMI, the main effect for month, $F(5, 54) = 6.43$, $p < .001$, indicated the scores varied across the months. The score for HMI was lower in July than in January and November. The main effect for group, $F(2, 58) = 3.59$, $p = .034$, indicated differing scores for hypomania ideation. Regardless of month, C-SADs scored lower than controls.

Summary of depression and behavioural symptoms. Depression and associated atypical vegetative behavioural symptoms were shown for the SAD groups in the autumn and winter months.

Sleep Quality and Food Preference Questionnaire. Mean self-rated scores and standard deviations for sleep quality are shown in Table 10.

Table 10

Means and Standard Deviations for Four Sleep Quality Scales: Good/ Bad Nights, Restful/ Disturbed Nights, Slept Lightly/ Slept Deeply, and Felt Refreshed/ Fatigued for Control, Subsyndromal SAD and Clinical SAD groups

	Group	January		March		May		July		September		November	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Good/ bad nights	Control	2.05	0.90	1.91	1.15	2.32	1.28	2.55	1.65	2.59	1.68	3.09	1.74
	S-SAD	2.76	1.18	3.05	1.43	3.57	1.78	3.29	1.71	3.19	1.50	3.05	1.91
	C-SAD	2.83	1.25	4.11	1.84	3.28	1.53	3.28	1.74	3.50	1.79	3.33	1.50
Restful/ disturbed nights	Control	2.41	1.44	2.05	1.29	2.50	1.50	2.50	1.74	2.27	1.42	2.91	1.74
	S-SAD	2.86	1.42	3.67	1.59	3.67	1.83	3.81	1.83	3.29	1.90	3.05	1.86
	C-SAD	2.94	1.66	4.06	1.95	3.33	1.46	3.22	1.73	3.72	1.87	3.44	1.72
Slept lightly/ deeply	Control	5.45	1.41	4.95	1.91	4.64	1.62	4.50	1.68	4.73	1.72	4.36	1.65
	S-SAD	4.81	1.54	4.24	1.76	4.29	1.35	4.52	1.50	4.10	1.76	5.14	1.65
	C-SAD	4.83	1.92	4.00	1.85	4.28	1.93	4.61	2.17	4.00	2.03	4.17	1.72
Felt refreshed/ fatigued	Control	2.95	1.36	2.77	1.54	3.41	1.59	3.27	1.91	3.23	1.74	3.32	1.76
	S-SAD	3.29	1.23	4.10	1.87	3.95	1.47	3.81	1.89	3.52	1.86	2.90	1.58
	C-SAD	3.06	1.66	4.33	1.68	4.28	1.87	4.28	2.05	3.94	1.89	3.94	1.83

Note. Ratings were on four 7-point scales where 1 = good nights, 7 = bad nights; 1 = restful nights, 7 = disturbed nights; 1 = slept lightly, 7 = slept deeply; and 1 = felt refreshed, 7 = felt fatigued.

n = 22 Control, 21 Subsyndromal SAD (S-SAD), and 18 Clinical SAD (C-SAD) participants.

For Sleep Quality, initial trend analyses using polynomial contrasts were performed separately for each of the four scales: good nights/ bad nights, restful nights/ disturbed nights, slept lightly/ slept deeply, and refreshed/ fatigued. For the refreshed/ fatigued scale, trend analyses revealed a significant quadratic main effect for month.

Repeated measures 6 x 3 (Month x Group) MANOVAs were also conducted for each of the four sleep quality scales. Overall, MANOVA revealed significant main effects for group for the good/ bad nights and restful/ disturbed nights scales, and a main effect for month for the felt refreshed/ fatigued scale only. There were no significant interactions.

From the MANOVA for the good/ bad nights scale, the main effect for group, $F(2, 58) = 5.71, p = .005$, indicated significant differences between the groups. Subsequent univariate analyses and post hoc pairwise comparisons showed both SAD groups to rate their sleep quality worse than controls.

From the MANOVA for the restful/ disturbed nights scale, the main effect for group, $F(2, 58) = 5.97, p = .004$, indicated significant differences between the groups. Subsequent univariate analyses and post hoc pairwise comparisons showed both SAD groups to have significantly more disturbed nights than controls.

Polynomial contrasts for refreshed/ fatigued nights revealed a significant quadratic effect for month, $F(1, 58) = 8.69, p = .005$, indicating participants overall showed a seasonal effect. Figure 9 shows the seasonal pattern reflecting a peak for

the autumn and winter months. Participants were more fatigued for autumn and winter than for spring and summer.

From the MANOVA for the refreshed/ fatigued scale, a main effect was revealed for Month, Pillai's Trace = .204, $F(5, 54) = 2.76$, $p = .027$, indicating significant differences across the months regardless of group. Subsequent post hoc pairwise comparisons showed participants to be significantly more fatigued for March, May, July, and September than for January.

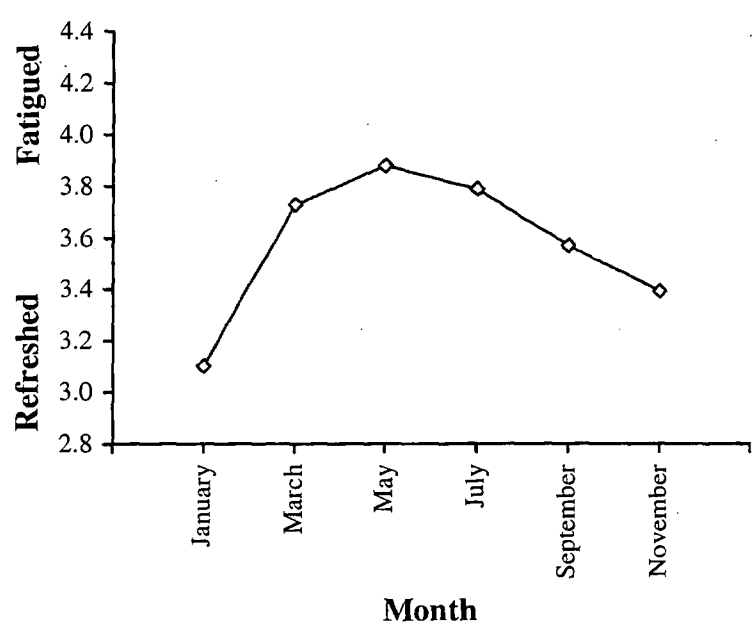


Figure 9. Seasonal pattern on sleep quality scale (felt refreshed/ fatigued) for participants overall across a twelve month period

For food preference, the percentage of each group experiencing a change in each of the four choices was calculated for each month, and is presented in Table 11. Higher percentages of the SAD groups are shown to prefer fats for the autumn

Table 11

Percentage of each Group (Control, Subsyndromal SAD, and Clinical SAD) Recording Changes in Food Preference (Fats, Protein, Carbohydrates) at Bimonthly Intervals

Preference	Group	January	March	May Percentage of Group	July	September	November
Fats	Control	4.35		8.7	4.35	4.35	
	S-SAD	4.76	14.28	33.35	19.05	4.76	9.5
	C-SAD	16.7	22.2	44.45	38.9	16.7	16.7
Protein	Control	4.35			8.7		
	S-SAD		14.28	9.5	4.76		9.5
	C-SAD				5.55		5.55
Carbohydrates	Control	13.05	8.7	4.35	8.7		4.35
	S-SAD	14.28	23.81	28.6	33.35	14.28	4.76
	C-SAD	33.35	22.2	44.45	44.45	33.35	38.9

Note . A missing value was possible, representing a particular group having no participants recording changes in food preference for the month.

n = 22 Control, 21 Subsyndromal SAD (S-SAD), and 18 Clinical SAD (C-SAD) participants.

and winter, than controls. Table 11 also shows the SAD groups had greater preference for carbohydrates than controls. Of the two SAD groups, a greater percentage of C-SADs recorded preferences for fats and carbohydrates than S-SADs.

Constitutional variables

Mean scores and standard deviations for body mass index (BMI), percentage body fat, systolic and diastolic blood pressure, sublingual temperature, finger and face skin temperature, and oxygen uptake are shown in Table 12.

Body mass index. Trend analyses using planned polynomial contrasts revealed no significant quadratic main effects of interactions. A 6 x 3 (Month x Group) repeated measures MANOVA was also conducted. MANOVA revealed a main effect for month only and no significant interactions. The main effect for month, Pillai's Trace = 2.68, $F(5, 54) = 2.68$, $p = .031$, indicating significant differences across the months. Table 12 shows these differences. Subsequent post hoc analyses show BMI to be significantly higher in July than in March and October. BMI is also higher in January than in March.

Percentage body fat. Trend analysis using planned polynomial contrasts and a 6 x 3 (Month x Group) repeated measures MANOVA were conducted. Neither the polynomial contrasts, nor the MANOVA revealed any significant main effects or interactions.

Table 12

Means and Standard Deviations for Body Mass Index, Percentage BodyFat, Systolic and Diastolic Blood Pressure and Sublingual Temperature for Control, Subsyndromal SAD, and Clinical SAD Groups at Bimonthly Testing Sessions

	Group	January		March		May		July		September		November	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Body Mass Index	Control	26.32	5.29	23.29	4.74	23.53	4.91	23.51	5.05	23.31	5.06	23.57	5.23
	S-SAD	24.12	3.85	23.92	3.71	23.94	3.47	24.08	3.52	24.19	3.68	24.07	3.86
	C-SAD	25.55	5.00	25.31	5.29	25.70	5.30	25.92	5.02	25.47	4.98	25.34	4.97
Percentage Body Fat	Control	27.10	8.13	26.43	8.75	26.48	9.33	26.29	9.40	26.28	9.79	26.61	8.91
	S-SAD	27.77	7.01	27.26	7.10	26.18	7.65	26.47	6.03	27.33	6.35	27.52	7.17
	C-SAD	31.18	6.13	30.17	5.63	30.33	6.55	30.92	5.83	30.96	5.82	30.91	6.86
Systolic Blood Pressure	Control	120.59	15.54	119.27	14.09	120.18	11.48	119.23	12.33	119.44	12.37	120.55	14.65
	S-SAD	115.43	15.28	121.33	12.31	120.67	14.20	121.43	14.02	122.81	16.69	121.90	15.58
	C-SAD	119.61	10.77	123.94	18.16	120.94	15.39	121.00	13.01	121.89	12.18	122.17	15.41
Diastolic Blood Pressure	Control	74.59	9.91	74.64	7.23	76.91	7.31	75.59	7.63	76.14	6.20	76.59	8.96
	S-SAD	75.29	10.03	77.33	10.94	78.52	10.06	76.90	9.66	80.86	10.94	78.67	8.56
	C-SAD	74.89	8.90	79.78	11.17	75.33	8.55	76.83	9.74	79.11	10.32	77.22	13.03
Sublingual Temperature	Control	36.60	0.33	36.49	0.39	36.58	0.46	36.53	0.41	36.61	0.41	36.56	0.41
	S-SAD	36.44	0.34	36.43	0.37	36.47	0.38	36.27	0.47	36.40	0.30	36.43	0.38
	C-SAD	36.52	0.32	36.39	0.39	36.46	0.31	36.48	0.33	36.47	0.28	36.42	0.67

Note . n = 23 Control, 22 Subsyndromal SAD, and 18 Clinical SAD participants

Blood pressure. Trend analyses using planned polynomial contrasts were conducted for systolic and diastolic blood pressure separately.

Polynomial contrasts revealed no significant quadratic or cubic main effects. Repeated measures 6 x 3 (Month x Group) MANOVAs were also conducted for systolic and diastolic measures separately, revealing a main effect for month for diastolic blood pressure only. There were no other main effects or interactions for systolic blood pressure. The main effect for month for diastolic blood pressure, Pillai's Trace = 2.87, $F(5, 54) = 2.87$, $p = .023$, indicated significant differences across the months which are shown in Table 12. Subsequent post hoc pairwise comparisons show diastolic blood pressure to be significantly higher in September, than in January or July, and also higher in March than in January.

Sublingual temperature. Trend analyses using planned polynomial contrasts revealed no significant quadratic main effects or interactions. A 6 x 3 (Month x Group) repeated measures MANOVA was conducted, revealing no significant main effects or interactions.

Oxygen uptake. Trend analysis using planned polynomial contrasts and a 6 x 3 (Month x Group) repeated measures MANOVA were conducted. Neither the polynomial contrasts, nor the MANOVA revealed any significant main effects or interactions.

Summary of results for constitutional measures. Body mass index showed a peak in July for all participants. Diastolic blood pressure showed a peak in March

and September for all participants. There were no other month or group differences for the constitutional variables.

Psychophysiological measures

Mean scores and standard deviations for actual and predicted RSA are presented in Table 13, and for HR and SCL in Table 14.

Actual respiratory sinus arrhythmia. For actual RSA, trend analyses using planned polynomial contrasts were performed separately for each condition, baseline and stressor. No significant quadratic or cubic polynomial contrasts were revealed for either the baseline or stressor condition.

A 6 x 2 x 3 (Month x Condition x Group) multivariate repeated measures MANOVA was also conducted for actual RSA. MANOVA revealed a significant main effect for condition, Pillai's Trace = .781, $F(1, 58) = 207.37$, $p < .001$. All participants had significantly lower actual RSA scores for the stressor condition than at baseline. No other significant main effects or interactions were revealed.

Predicted respiratory sinus arrhythmia. Trend analyses using planned polynomial contrasts were conducted for each condition, baseline and stressor, separately. Polynomial contrasts revealed a significant quadratic effect for month for the baseline condition, $F(1, 58) = 6.57$, $p = .013$, indicating seasonal variation across the months. Figure 10 shows this seasonal effect for the baseline condition of predicted RSA to peak for March, May, July, and September, as compared to January.

Table 13

Means and Standard Deviations for Actual and Predicted Respiratory Sinus Arrhythmia for Control, Subsyndromal SAD, and Clinical SAD Groups at Bimonthly Testing Sessions

Condition	Group	January		March		May		July		September		November	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Actual Respiratory Sinus Arrhythmia													
Baseline	Control	12.55	6.91	12.51	5.95	14.02	5.34	15.30	6.11	14.97	7.13	12.62	6.11
	S-SAD	12.26	6.46	11.68	6.11	12.80	7.28	12.14	7.03	11.92	6.08	12.65	5.85
	C-SAD	12.39	6.14	12.90	5.53	13.21	6.89	13.05	7.61	14.03	7.74	12.80	5.47
Stressor	Control	6.35	2.59	6.41	3.16	5.71	2.46	6.49	2.66	5.87	3.23	6.14	2.61
	S-SAD	5.70	2.29	5.40	2.11	6.14	3.19	5.48	2.92	4.77	1.46	5.48	2.34
	C-SAD	5.27	2.38	5.26	2.31	6.10	3.12	6.15	3.41	5.43	2.98	5.11	2.61
Predicted Respiratory Sinus Arrhythmia													
Baseline	Control	11.67	6.27	13.17	5.30	13.27	5.39	13.39	4.58	13.23	4.49	12.10	4.83
	S-SAD	10.39	4.55	10.91	5.60	12.10	6.73	11.60	6.02	11.34	4.97	11.12	4.70
	C-SAD	11.30	5.91	12.34	5.56	11.81	6.73	12.11	7.07	12.83	5.69	11.87	5.38
Stressor	Control	6.92	4.44	6.43	3.35	5.85	4.30	7.17	3.73	6.91	3.49	5.93	4.20
	S-SAD	6.32	3.08	6.70	3.38	6.77	4.44	6.58	4.10	5.78	2.80	5.89	3.13
	C-SAD	6.90	4.04	7.16	3.50	7.14	5.19	7.66	5.91	7.74	4.90	6.13	3.00

Note . n = 22 Control, 21 Subsyndromal SAD (S-SAD), and 18 Clinical SAD (C-SAD) participants.

Table 14

Means and Standard Deviations for Heart Rate (bpm) and Skin Conductance Level (MicroSiemens) for Control, Subsyndromal SAD, and Clinical SAD groups at Bimonthly Testing Sessions

Condition	Group	January		March		May		July		September		November	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Heart Rate													
Baseline	Control	77.96	9.40	74.54	10.47	74.72	9.35	76.64	8.74	74.55	11.08	77.23	10.89
	S-SAD	74.46	9.06	72.58	11.17	73.10	10.35	71.36	11.36	76.41	12.74	77.71	9.45
	C-SAD	69.26	18.41	72.49	10.26	71.26	10.80	74.51	10.36	73.27	8.20	75.14	13.10
Stressor	Control	82.27	10.14	78.16	8.86	79.40	7.99	79.30	8.90	78.44	10.92	81.25	9.08
	S-SAD	78.30	9.91	75.50	10.57	77.69	11.92	75.16	11.59	78.89	12.80	80.95	10.33
	C-SAD	77.00	9.97	78.90	11.24	78.42	9.67	79.71	8.41	78.01	8.51	80.38	13.10
Skin conductance level													
Baseline	Control	6.36	4.02	6.91	5.10	9.64	6.10	8.40	4.17	7.80	4.71	7.12	5.21
	S-SAD	6.08	3.98	7.77	4.48	6.65	8.50	7.88	4.98	8.27	5.07	7.34	4.40
	C-SAD	6.21	4.21	9.49	7.54	7.89	5.60	7.20	6.11	7.13	3.96	10.27	8.97
Stressor	Control	7.32	4.19	8.21	5.24	9.36	4.16	9.58	3.94	8.70	4.62	8.46	5.44
	S-SAD	7.57	3.92	8.97	4.54	9.59	8.68	8.96	5.19	9.28	4.93	8.34	4.31
	C-SAD	7.51	4.34	10.97	8.25	9.81	5.94	8.77	7.00	8.66	4.45	11.57	9.14

Note . n = 22 Control, 21 Subsyndromal SAD (S-SAD), and 18 Clinical SAD (C-SAD) participants.

For predicted RSA, a 6 x 2 x 3 (Month x Condition x Group) multivariate repeated measures MANOVA was also conducted. MANOVA revealed a significant main effect for condition, Pillai's Trace = .861, $F(1, 58) = 359.18$, $p < .001$, a significant interaction between month and condition, Pillai's Trace = .283, $F(5, 54) = 4.27$, $p = .002$. The interaction indicated baseline and stressor conditions varied across the months. Post hoc analyses between baseline and stressor conditions at each month show significant differences for all six comparisons at the $\alpha = .001$ significance level. The predicted RSA score for the stressor condition was significantly lower than at baseline in all months. Univariate repeated measures analyses and post hoc pairwise comparisons were also conducted revealing significant differences for the baseline condition only. Predicted RSA scores were significantly higher for March, May, July, and September, than for January (see Figure 10).

To identify the source of the significant main effects and interaction for predicted RSA, 6 x 3 (Month x Group) repeated measures MANOVAs were conducted separately using intercept and slope scores from individual linear regression analyses as dependent variables. No significant main effects or interactions were revealed either for the intercept or the slope values.

Respiratory sinus arrhythmia difference score. An RSA difference score was calculated to index RSA by subtracting predicted RSA from actual RSA due to the stressor corrected for changes in respiratory rate. Trend analyses using planned polynomial contrasts were performed separately for each condition, baseline and stressor. No significant quadratic or cubic polynomial contrasts were revealed for either the baseline or stressor condition of the RSA difference score. A 6 x 2 x 3

(Month x Condition x Group) multivariate repeated measures MANOVA was also conducted for the RSA difference score revealing no significant main effects or interactions.

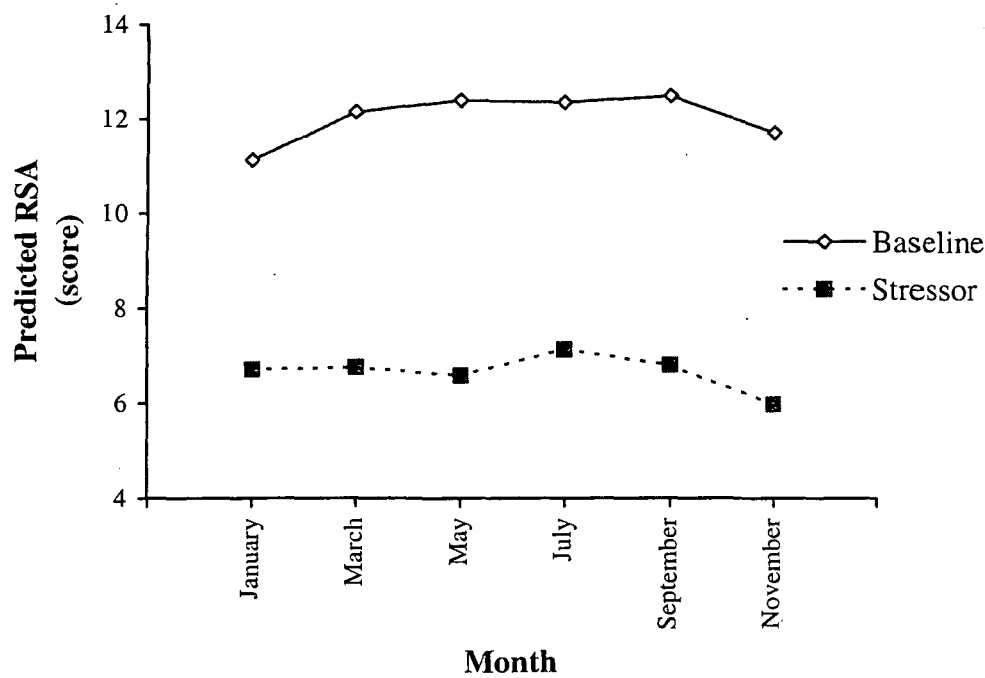


Figure 10. Mean predicted respiratory sinus arrhythmia scores at baseline and stressor condition at bimonthly testing sessions

Heart Rate. Initial analyses conducted were trend analyses using planned polynomial contrasts for each condition, baseline and stressor, separately. Planned polynomial contrasts showed no significant quadratic main effects or interactions, for either baseline or stressor condition.

For HR, a 6 x 2 x 3 (Month x Condition x Group) multivariate repeated measures MANOVA was also performed. Overall, HR revealed a main effect for condition, and significant two-way interactions between group and condition, and between month and condition.

The main effect for condition, Pillai's Trace = .697, $F(1, 58) = 133.58$, $p < .001$, indicated a significant difference between baseline and stressor conditions for participants overall. The significant interaction between group and condition, Pillai's Trace = .126, $F(2, 58) = 4.17$, $p = .02$, indicated that the groups varied between baseline and stressor conditions. Post hoc analyses between the conditions for each group showed HR to be significantly higher than baseline for each group.

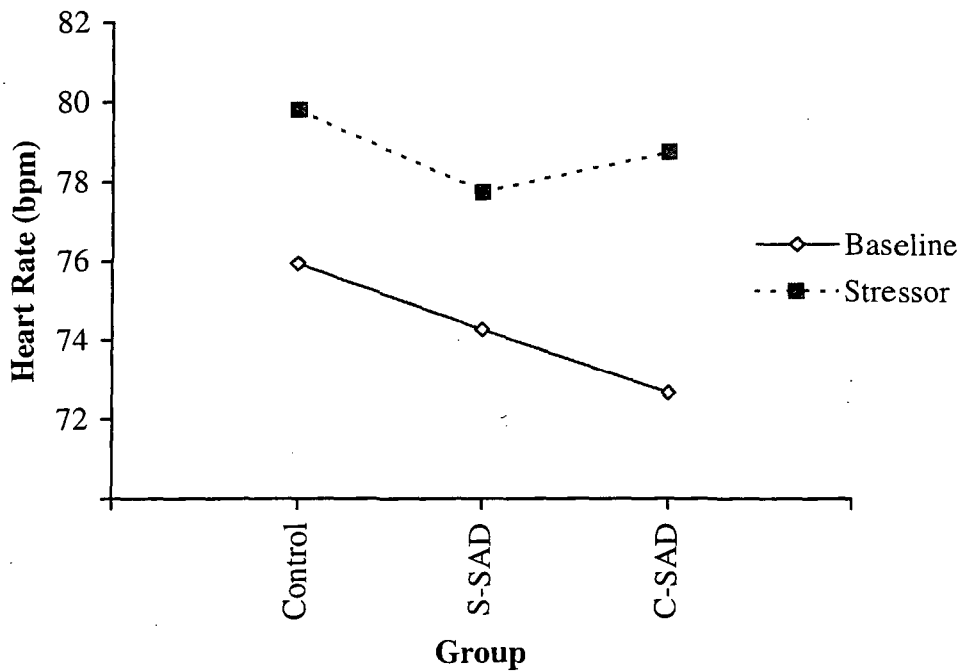


Figure 11. Mean heart rate (bpm.) at baseline and stressor levels for control, subsyndromal SAD (S-SAD) and clinical SAD (C-SAD) groups

Univariate analyses between the groups for each condition separately revealed no significant differences. To identify the source of interaction, HR difference scores were computed by subtracting baseline from stressor, and analysed as a between groups ANOVA. ANOVA revealed a main effect for group, $F(2, 58)$

= 3.64, $p = .03$. Figure 11 shows the difference score for C-SADs to be significantly greater than for S-SADs and controls.

The significant interaction between month and condition, Pillai's Trace = .188, $F(5, 54) = 2.50$, $p = .042$, indicated that HR varied across the months between baseline and stressor conditions. Post hoc analyses between the conditions for each month revealed significant differences for all months. HR was significantly higher for the stressor condition than baseline in each month at the $\alpha = .001$ significance level. Univariate analyses for each condition across the months separately revealed significant differences for the baseline condition only. Figure 12 shows baseline HR to be significantly lower in March and May than in November at the $p = .01$ significance level.

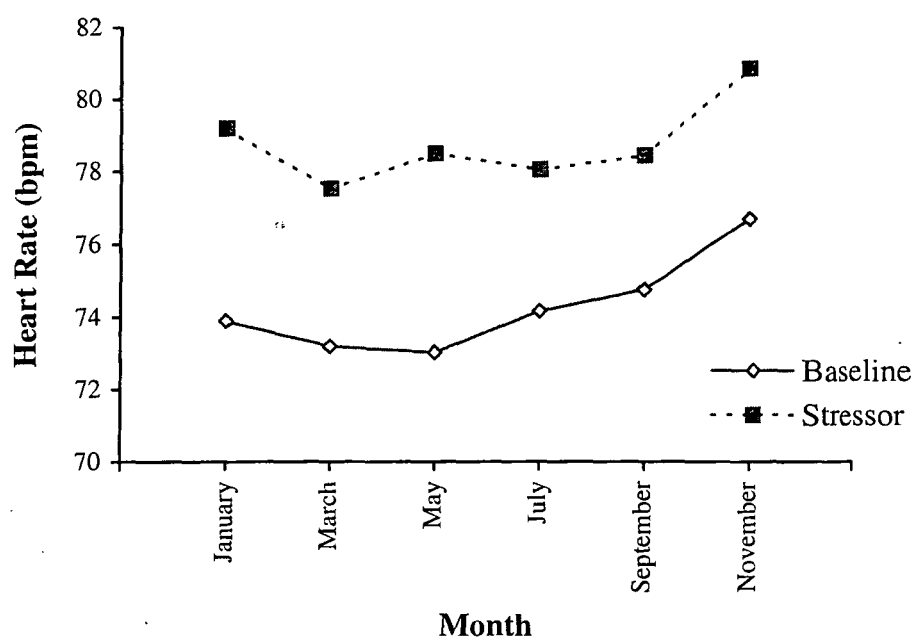


Figure 12. Mean heart rate (bpm) at baseline and stressor levels for participants overall at six bimonthly intervals

Skin Conductance Level. Initial analyses were trend analyses using planned polynomial contrasts conducted for baseline and stressor conditions separately revealing a significant cubic main effect for month for the stressor condition. A strong tendency towards significance was also revealed for cubic interactions between month and group for both baseline and stressor conditions.

For the baseline condition, the tendency towards significance for the cubic interaction between month and group, $F(2, 56) = 3.14, p = .051$, indicated each group differed in their seasonal pattern for SCL. Subsequent polynomial contrasts for each group separately revealed a significant quadratic main effect for month for controls, and a significant cubic effect for C-SADs. Figure 13 shows controls to peak for May and July, as compared to all other months. Figure 13 also shows C-SADs to peak for the autumn months, as compared to July and September. A secondary peak is also shown for November.

For the stressor condition, the significant cubic main effect for month, $F(1, 56) = 4.80, p = .033$, indicated that regardless of group, participants showed a seasonal pattern in SCL across the twelve-month period. The tendency towards significance for the cubic interaction between month and group, $F(2, 56) = 3.03, p = .056$, was due to a significant cubic effect for month for the C-SAD group only. Figure 13 shows the C-SAD group to peak for the stressor condition in the autumn months, and also in November.

For SCL, a 6 x 2 x 3 multivariate repeated measures MANOVA was also conducted. MANOVA revealed a main effect for condition, Pillai's Trace = .683, $F(1, 56) = 120.66, p < .001$. A strong tendency revealed for a three-way interaction between month, condition, and group, Pillai's Trace = .302, $F(10, 106) = 1.88, p = .055$, was due to significant effects for controls only for month in both baseline and stressor conditions. At baseline, controls had a significantly higher SCL in May as compared to January and March. For the stressor level, controls were significantly higher for May and July than for January.

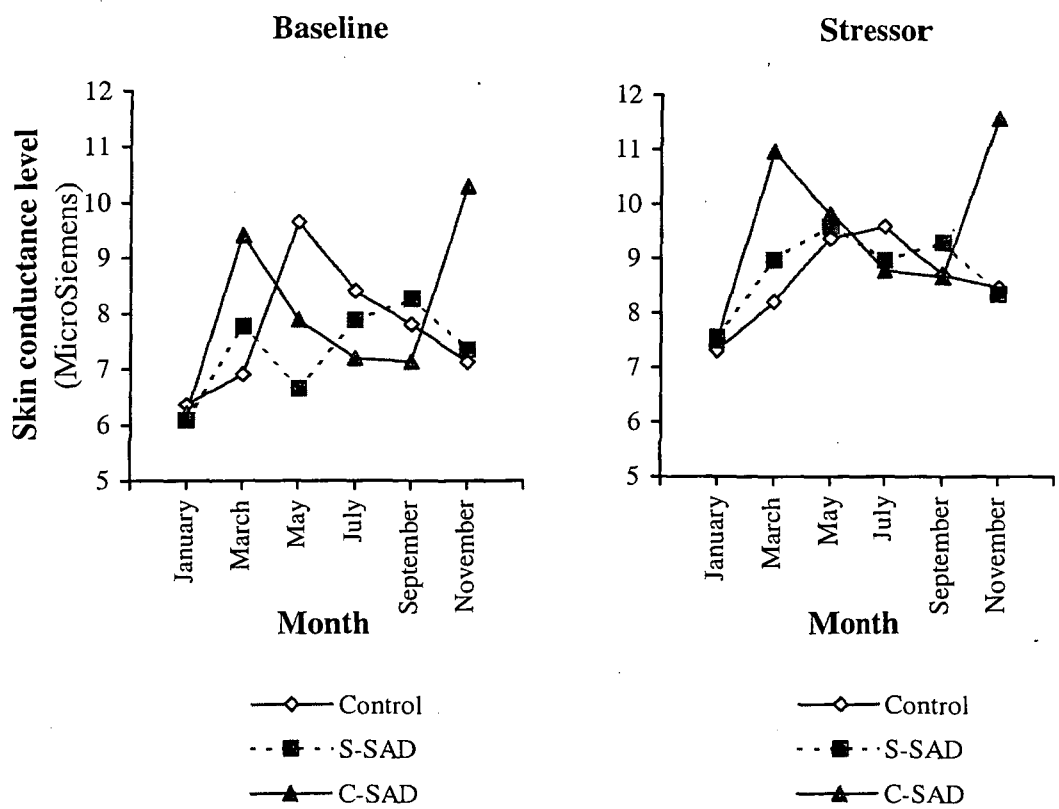


Figure 13. Seasonal pattern for Skin Conductance Level (μS) at baseline and stressor levels for control, subsyndromal SAD (S-SAD) and clinical SAD (C-SAD) groups

Means and standard deviations for pulse height are presented in Table 15, and for skin temperature are in Table 16.

Pulse height Percentage. A trend analysis using planned polynomial contrasts was conducted. No significant effects were revealed from the trend analysis. A 6 x 3 (Month x Group) repeated measures MANOVA was also conducted for pulse height. No significant main effects or interactions were revealed from the MANOVA.

Table 15
Means and Standard Deviations for Pulse Height Responses for Control, Subsyndromal SAD (S-SAD), and Clinical SAD (C-SAD) Groups at Six Bimonthly Testing Sessions

	Control		S-SAD		C-SAD	
	Mean	SD	Mean	SD	Mean	SD
January	113.06	52.36	111.61	56.46	109.78	51.08
March	83.93	30.62	116.43	72.2	90.63	54.63
May	101.38	37.17	118.6	57.95	98.21	33.82
July	131.94	87.46	104.2	52.92	112.11	59.63
September	106.40	54.55	114.21	47.16	102.83	34.58
November	110.47	46.64	100.40	31.52	89.45	17.83

Note. Pulse height was calculated as a percentage of baseline.
n = 23 control, 20 S-SAD, 18 C-SAD

Table 16

Means and Standard Deviations for Skin Temperature at Two Sites (Finger and Face) for Control, Subsyndromal SAD, and Clinical SAD Groups at Bimonthly Testing Sessions

Site	Condition	Group	January		March		May		July		September		November	
			Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Finger	Baseline	Control	33.65	2.01	32.25	4.22	31.39	3.95	30.51	4.88	30.81	4.51	32.36	4.58
		S-SAD	34.45	1.04	31.92	4.98	30.19	4.98	30.16	5.19	31.21	4.68	33.41	3.5
		C-SAD	33.48	3.31	31.18	3.43	31.23	4.12	30.48	4.35	31.33	4.46	33.56	1.42
	Stressor	Control	33.71	1.46	31.47	3.99	31.91	3.41	31.16	4.20	31.02	3.30	31.68	4.52
		S-SAD	34.15	1.14	31.56	4.44	29.85	5.00	30.02	4.96	31.87	3.22	33.10	3.54
		C-SAD	33.25	3.14	31.36	2.02	30.75	3.88	31.16	3.46	30.92	4.48	32.54	1.99
Face	Baseline	Control	32.31	1.65	32.69	1.24	32.46	1.67	31.06	3.18	32.51	1.42	32.56	1.62
		S-SAD	32.44	0.93	31.92	1.30	32.16	1.55	31.44	1.55	32.62	1.64	32.47	1.75
		C-SAD	32.40	1.15	31.56	1.81	31.99	1.12	31.84	1.78	31.66	1.67	32.27	1.33
	Stressor	Control	33.31	1.38	33.18	1.22	33.49	1.33	33.06	1.51	33.39	1.25	33.63	1.32
		S-SAD	33.08	1.19	32.22	1.17	33.40	1.16	32.98	1.46	33.83	1.27	33.88	1.34
		C-SAD	33.30	1.01	32.81	1.30	32.94	1.18	32.68	1.48	32.95	1.47	33.38	1.25

Note. For finger skin temperature (N = 51; 17 control, 17 S-SAD, and 17 C-SAD participants)

For face skin temperature (N = 47; 16 control, 17 S-SAD, and 14 C-SAD participants)

Skin temperature. For finger skin temperature, trend analyses using planned polynomial contrasts were conducted for baseline and stressor separately.

Polynomial contrasts revealed a significant quadratic effect for month for both baseline and stressor conditions. The significant quadratic effects for month for the baseline condition, $F(1, 55) = 32.63, p < .001$, and for the stressor condition, $F(1, 48) = 31.29, p < .001$, indicated seasonal patterns for each condition. Figure 14 shows finger skin temperature to reach a low value for May, July, and September.

A $6 \times 2 \times 3$ (Month \times Condition \times Group) repeated measures MANOVA was also conducted for finger skin temperature. MANOVA revealed a main effect for month, and a significant interaction between month and condition. The main effect for month, Pillai's Trace = .518, $F(5, 44) = 9.46, p < .001$, indicated that finger skin temperature varied across the months. The significant Month \times Condition interaction, Pillai's Trace = .225, $F(5, 44) = 2.56, p = .041$, indicated that baseline and stressor conditions varied across the months. Subsequent post hoc analyses between baseline and stressor for each month revealed significant differences for November only. Finger skin temperature was significantly higher for baseline than for the stressor condition in November.

Univariate repeated measures ANOVAs were also conducted for baseline and stressor conditions separately. ANOVA revealed significant main effects for month for both baseline and stressor conditions. For baseline, finger skin temperature was significantly lower for May, July, and September, than for January at the $p = .001$ significance level. Finger temperature was also significantly lower for March than for January at the $p = .01$ significance level. For the stressor condition, finger skin

temperature was significantly lower for March, May, July, and September, than January at the $p = .001$ significance level. Figure 14 shows these differences for finger skin temperature.

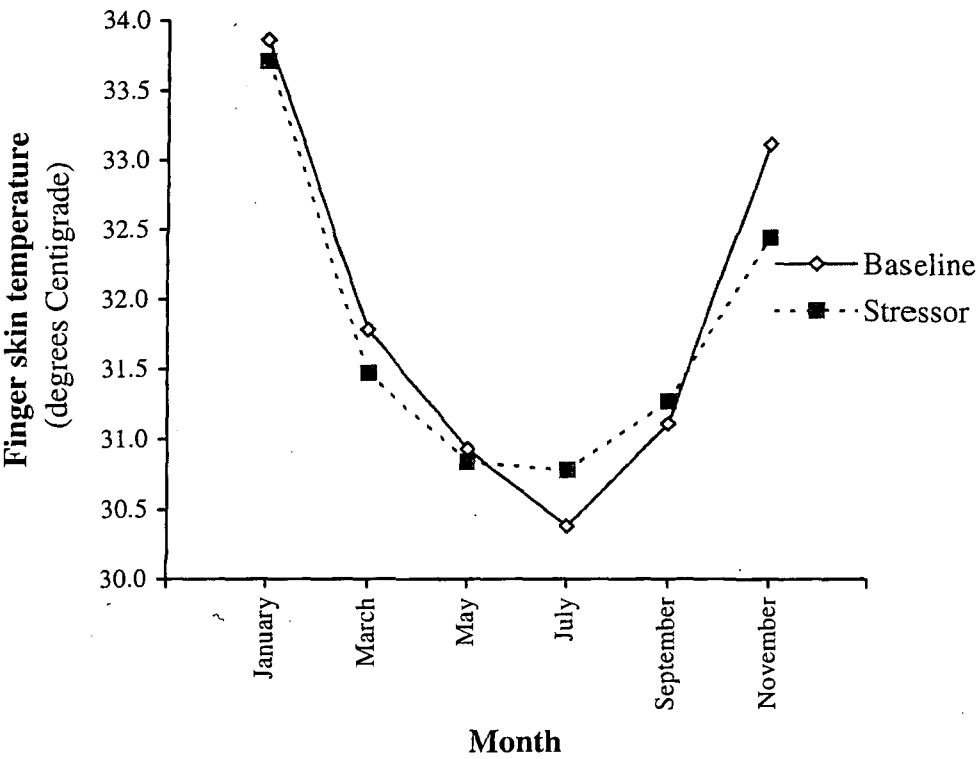


Figure 14. Mean finger skin temperature at baseline and stressor conditions for participants overall in bimonthly testing sessions

For face skin temperature, trend analyses using planned polynomial contrasts were conducted for baseline and stressor separately. Polynomial contrasts revealed significant quadratic effect for month for both baseline and stressor conditions. The significant quadratic effect at baseline, $F(1, 53) = 5.48, p = .023$, and for the stressor condition, $F(1, 44) = 5.67, p = .022$, indicated a seasonal pattern. Figure 15 shows face skin temperature to be lower for July as compared to all other months.

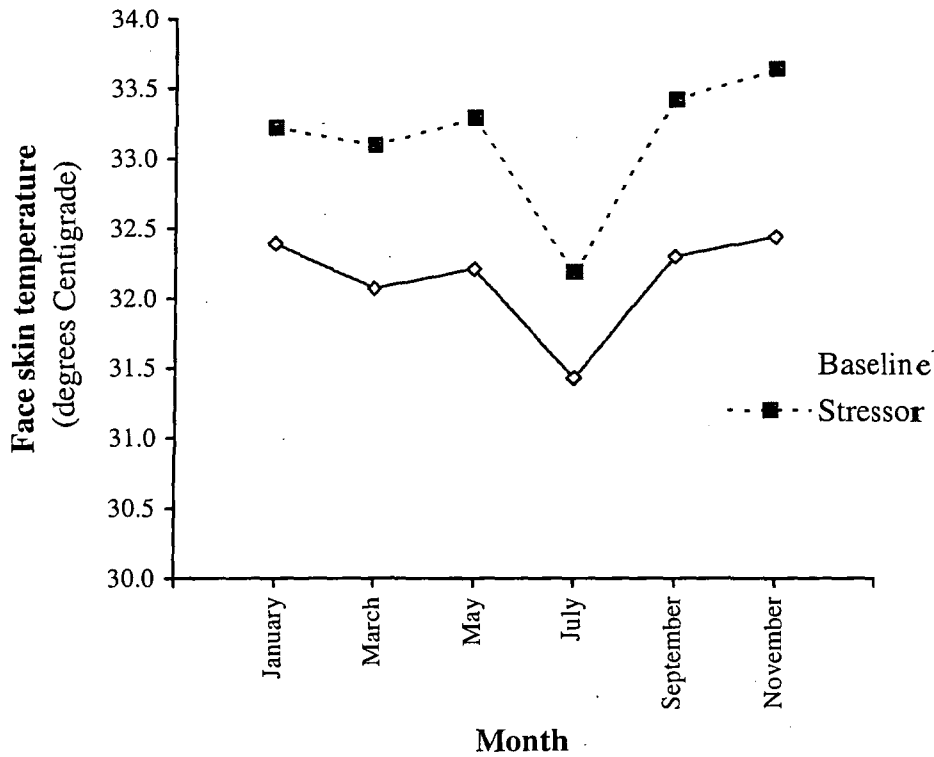


Figure 15. Mean face skin temperature at baseline and stressor conditions for participants overall in bimonthly testing sessions

A 6 x 2 x 3 (Month x Condition x Group) repeated measures MANOVA was also conducted for face skin temperature. MANOVA revealed main effects for month, and for condition. The main effect for month, Pillai's Trace = .289, $F(5, 40) = 3.25$, $p = .015$, indicated that regardless of group or condition, participants varied across the months. Face skin temperature was significantly lower for July, than for all other months except March. The main effect for condition, Pillai's Trace = .876, $F(1, 44) = 311.45$, $p < .001$, indicated a significant difference between the conditions. Face skin temperature was significantly higher for the stressor condition than at baseline.

Stressor task. Means and standard deviations for two conditions; addition and subtraction of the mental arithmetic stressor task (response time) are presented in Table 17.

Trend analyses using planned polynomial contrasts were conducted for each condition; addition and subtraction separately. No significant quadratic or cubic main effects or interactions were revealed for either the addition or subtraction condition.

Multivariate 6 x 3 (Month x Group) repeated measures MANOVAs were also conducted for each condition separately. No significant main effects or interactions were revealed for the addition condition. For the subtraction condition, MANOVA revealed a main effect for month, Pillai's Trace = .196, $F(5, 51) = 2.48$, $p = .043$. The response time for mental arithmetic was significantly longer in July than in January, March, and September. The reaction time was also significantly longer in November than in January.

Table 17

Means and Standard Deviations (Response Time) for Addition and Subtraction Conditions of the Mental Arithmetic Stressor Task by Control, Subsyndromal SAD (S-SAD), and Clinical SAD (C-SAD) Groups at Each of Six Bimonthly Testing Sessions

Condition	Group	January		March		May		July		September		November	
		Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>
Response time (ms.)													
Addition	Control	3607.28	513.91	3721.12	434.92	3664.19	365.68	3845.45	254.1	3746.12	353.29	3745.34	378.28
	S-SAD	3723.2	477.72	3729.26	426.67	3938.28	407.59	3689.27	353.49	3678.4	493.23	3747.17	425.01
	C-SAD	3586.63	519.16	3680.03	469.86	3723.41	381.47	3544.2	538.65	3603.26	511.80	3529.33	476.14
Subtraction	Control	3494.51	371.46	3564.73	373.31	3612.23	309.26	3717.08	367.82	3579.37	368.07	3698.69	348.48
	S-SAD	3707.76	508.79	3700.13	407.73	3764.61	406.96	3815.77	392.20	3689.14	542.00	3761.33	487.07
	C-SAD	3576.08	492.93	3603.11	486.94	3660.53	364.28	3659.02	411.52	3543.19	369.07	3595.72	431.86

Note. Mental arithmetic task consisted 60 multiple choice trials: 32 addition and 28 subtraction.

n = 22 control, 21 S-SAD, and 18 C-SAD

Summary of results for psychophysiological measures. Actual and predicted RSA were shown to decrease, and HR and SCL to increase from baseline to stressor condition. For HR, this increase from baseline to stressor was greater for C-SAD than for S-SADs and controls. For SCL, controls showed a peak in May as compared to January. For the mental arithmetic task, the subtraction condition was shown to have a longer reaction time in July as compared to January, March, and September. Face skin temperature increased from baseline to stressor. Finger skin temperature showed a decrease from baseline to stressor conditions for November only.

Several variables showed seasonal variation for participants overall. The baseline condition of predicted RSA showed a peak in autumn/ winter. The SCL stressor condition also showed a peak in autumn. Finger and face skin temperature each showed a trough in July for both baseline and stressor conditions.

Emotion analyses

For the trait version of the TESI, mean self-rated scores and standard deviations for Tension Stress felt (Part A), and Effort Stress expended (Part B), from four sources (work, family, finance, and own body) are presented in Tables 18 and 19 respectively.

Table 18

Means and Standard Deviations for Tension Stress Felt from Four Stressors (Work, Family, Finance, and Body) from the Tension and Effort Stress Inventory for Control, Subsyndromal SAD (S-SAD), and Clinical SAD (C-SAD) Groups in the Thirty Days Prior to each of Six Bimonthly Testing Sessions

Stressor	Group	January		March		May		July		September		November	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Work	Control	2.91	1.87	3.27	1.70	4.09	1.90	2.59	1.71	3.77	1.95	4.59	2.38
	S-SAD	3.86	2.08	4.52	1.91	4.14	1.98	3.52	1.63	4.24	2.05	4.19	1.81
	C-SAD	2.89	1.60	4.67	1.37	3.83	1.89	3.28	1.60	5.17	1.62	4.67	1.91
Family	Control	1.82	1.26	2.45	1.74	2.18	1.5	2.14	1.52	2.00	1.54	2.09	1.27
	S-SAD	4.14	1.82	3.95	1.83	3.62	1.4	4.05	1.69	3.43	1.6	4.10	1.87
	C-SAD	3.50	1.62	3.61	1.61	3.78	2.02	3.56	1.65	3.28	1.53	4.00	1.81
Finance	Control	3.09	2.00	3.00	1.69	2.86	1.55	2.68	1.59	3.09	1.57	3.09	1.74
	S-SAD	3.76	1.92	3.95	1.75	3.76	2.00	4.10	1.58	4.05	1.77	3.29	1.93
	C-SAD	4.78	1.86	4.61	1.75	4.39	2.35	4.39	2.03	4.00	2.09	4.61	1.85
Body	Control	2.41	1.53	2.55	1.74	2.05	1.50	2.59	1.56	2.59	1.74	2.18	1.47
	S-SAD	3.90	1.61	3.62	1.50	3.76	1.67	4.19	1.81	4.43	1.80	3.81	1.5
	C-SAD	3.06	1.83	3.89	1.81	4.06	1.47	4.22	1.35	3.89	2.00	3.94	2.15

Note. Tension stress ratings were made on a 7-point scale (1 = not at all, 7 = very much)

n = 22 Control, 21 S-SAD, and 18 C-SAD participants.

Table 19

Means and Standard Deviations for Effort Stress Expended for Four Stressors (Work, Family, Finance, and Body) from the Tension and Effort Stress Inventory for Control, Subsyndromal SAD, and Clinical SAD Groups in the Thirty Days Prior to each of Six Bimonthly Testing Sessions

Stressor	Group	January		March		May		July		September		November	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Work	Control	3.32	2.12	3.95	1.81	4.09	2.24	2.73	1.91	3.82	1.74	4.64	2.22
	S-SAD	4.24	2.12	4.90	1.84	4.33	1.83	3.95	1.86	4.48	1.72	4.48	1.86
	C-SAD	3.56	1.89	4.33	1.53	3.89	1.71	3.44	1.79	5.33	1.57	4.94	1.83
Family	Control	2.14	1.67	2.55	2.02	2.59	1.92	2.05	1.36	1.95	1.33	2.41	1.65
	S-SAD	4.00	1.76	4.19	1.72	3.76	1.45	4.14	1.71	4.19	1.50	4.71	1.62
	C-SAD	3.50	1.82	3.61	1.91	3.67	1.71	3.44	1.69	3.11	1.64	4.44	1.85
Finance	Control	2.95	2.08	2.91	1.82	2.50	1.90	2.27	1.42	2.59	1.53	2.77	1.66
	S-SAD	3.76	1.89	3.76	1.87	3.00	1.41	3.10	1.76	3.76	1.64	3.86	1.93
	C-SAD	4.17	1.95	4.17	1.86	4.28	2.14	3.72	1.84	3.44	1.98	3.67	1.97
Body	Control	2.00	1.41	2.59	1.71	1.86	1.39	2.36	1.65	2.64	2.04	1.91	1.48
	S-SAD	3.67	1.80	4.24	1.70	3.38	1.63	3.33	1.68	4.00	1.67	3.52	1.44
	C-SAD	2.94	1.63	4.06	1.92	4.06	1.95	4.06	1.80	3.94	1.80	3.61	1.75

Note. Effort stress ratings were made on a 7-point scale (1 = not at all, 7 = very much)

n = 22 Control, 21 S-SAD, and 18 C-SAD participants.

Initial trend analyses using planned polynomial contrasts were conducted for each of the four stress sources for Tension Stress separately. For Tension stress felt planned polynomial contrasts revealed a significant cubic main effect for work stress, a significant quadratic main effect for body stress, and a significant cubic interaction between month and group for body stress.

For Tension Stress, the significant cubic main effect for work, $F(1, 58) = 10.22, p = .002$, indicated a seasonal pattern across the months for participants overall. Figure 16 shows peaks for felt tension stress from work in March/ May and September/ November.

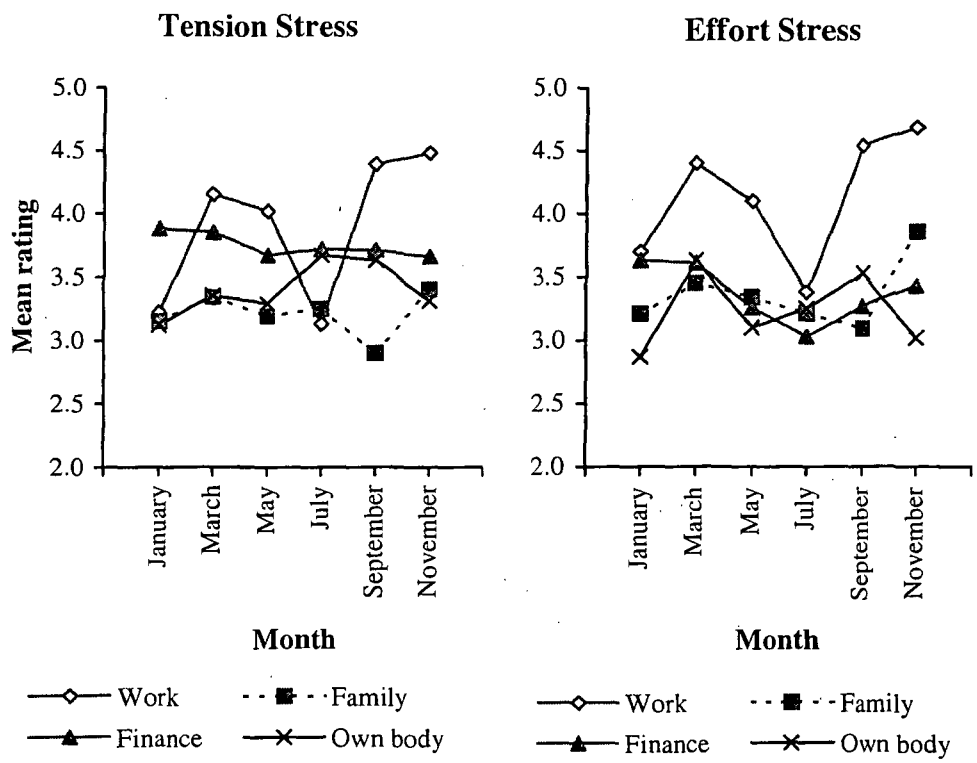


Figure 16. Degree of Tension Stress felt and Effort Stress for four sources of stress (Work, Family, Finance, and Body) in the thirty days prior to each testing session.

For body stress, the significant quadratic main effect, $F(1, 58) = 4.33, p = .042$, and the cubic interaction, $F(2, 58) = 3.22, p = .047$, indicated a significant seasonal effect which varied between the groups. Subsequent polynomials on body stress for each group revealed a significant cubic effect for S-SADs, and a significant quadratic effect for C-SADs. For S-SADs Figure 17 shows a trough in March/ May, and a peak in September. Figure 17 also shows a peak for C-SADs in March, May, and July.

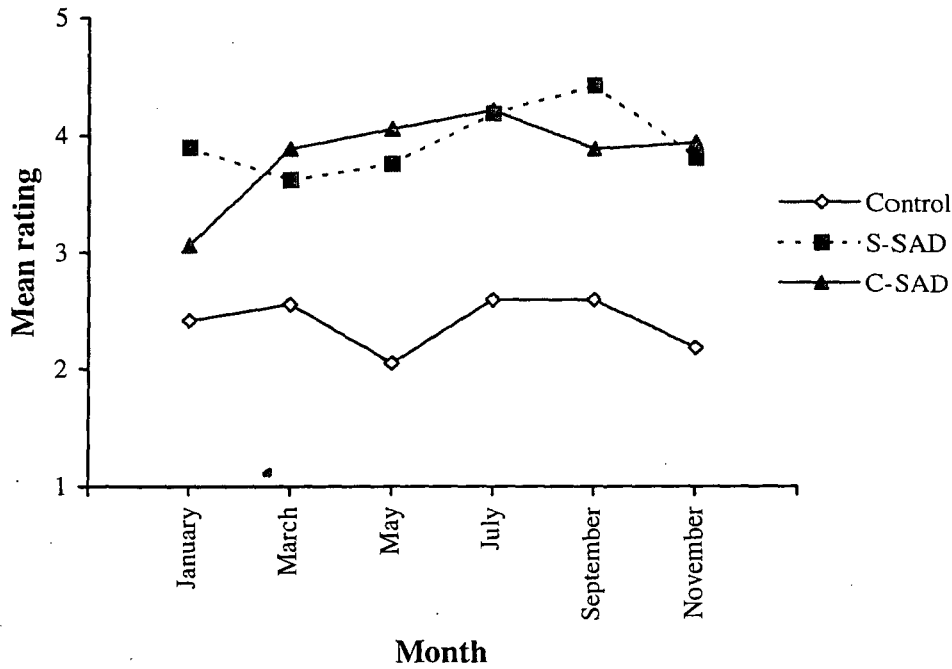


Figure 17. Degree of felt Tension Stress from own body shown by control, subsyndromal SAD (S-SAD) and clinical (C-SAD) Groups

Repeated measures 6 x 4 x 3 (Month x Stress Source: work/ family/ finance/ own body x Group) ANOVAs were also conducted for tension stress. Overall, for Tension Stress, ANOVA revealed significant main effects for stress source, and for

group, and significant Stress Source x Group, and Month x Stress Source interactions.

The main effects for stress source for tension stress, $F(2.73, 158.18) = 5.57$, $p = .002$, indicated significant differences in ratings between the stress sources. The main effect for group for tension stress, $F(2, 58) = 15.13$, $p < .001$, indicated significant differences between the groups. The significant interaction between stress source and group for tension ratings for tension stress felt between the sources of stress. Subsequent univariate analyses between the groups for each stress source revealed significant differences for family, finance, and body. Figure 18 shows these differences. S-SADs and C-SADs rated tension stress from family and body significantly higher than controls. For Tension Stress from finance, C-SADs rated higher, while S-SADs showed a strong tendency to rate higher than controls. The groups did not differ significantly for Tension Stress felt for work. Univariate repeated measures analyses for each group across the stress sources revealed significant differences for controls in Tension Stress felt. Controls felt tension stress more from work than from family and body, and also felt more tension stress from finance than family. The groups did not differ significantly for Tension Stress felt for work. Controls put up more effort for work than for family, finance, and body. Neither S-SADs, nor C-SADs, showed significant differences between the stress sources for tension felt.

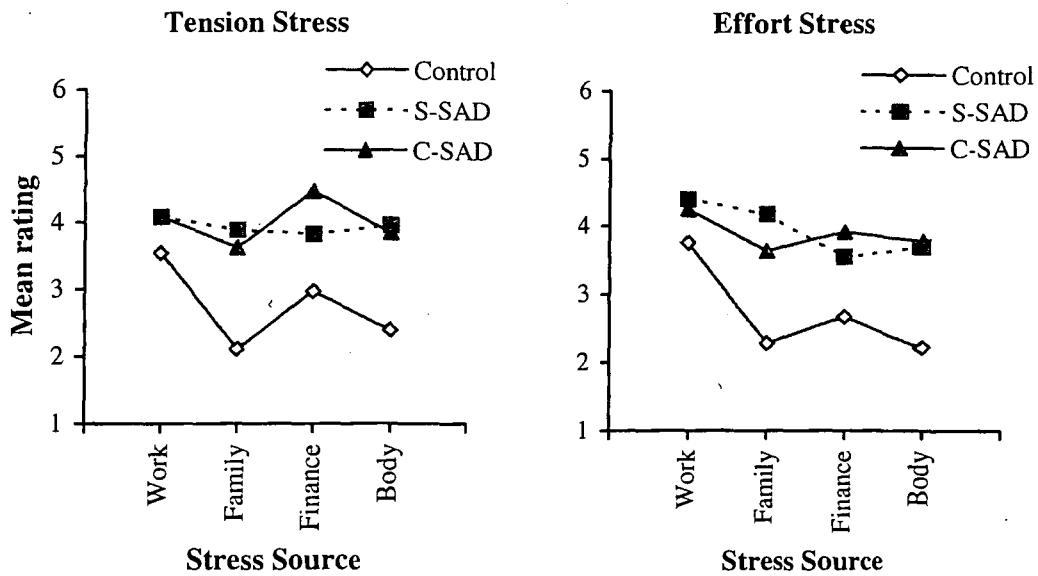


Figure 18. Degree of Tension Stress and Effort Stress for four sources of stress (Work, Family, Finance, and Body) shown by Control, Subsyndromal SAD (S-SAD) and Clinical SAD (C-SAD) Groups

The significant interactions between month and stress source for Tension Stress, $F(10.17, 589.81) = 4.36, p < .001$, indicated ratings for the stress sources varied across the months. Subsequent univariate repeated measures for each stress source across the months revealed significant differences in tension stress from work. Participants overall feel more stress from work in March, May, September, and November than in January, and July (Figure 16).

For Effort Stress, planned polynomial contrasts revealed significant cubic main effects for work, and for family, and a significant quadratic main effect for body. For Effort Stress, the significant cubic main effects for work, $F(1, 58) = 9.23$,

$p = .004$, and for family, $F(1, 58) = 7.31$, $p = .009$, indicated a seasonal pattern across the months for groups overall. The significant quadratic main effect for body, $F(1, 58) = 4.64$, $p = .035$, also indicated a seasonal pattern across the months for groups overall. Figure 16 shows a peak for effort stress related to work and family in March and November.

Repeated measures $6 \times 4 \times 3$ (Month x Stress Source: work/ family/ finance/ own body x Group) ANOVAs were also conducted for Effort Stress. Overall, for Effort Stress, ANOVA revealed significant main effects for stress source, for month, and for group, and significant Stress Source x Group, and Month x Stress Source interactions.

The main effect for month for Effort Stress, $F(4.59, 265.99) = 4.94$, $p < .001$, indicated effort stress varied across the months. The main effect for stress source for Effort Stress, $F(2.72, 157.57) = 10.65$, $p < .001$, indicated significant differences in ratings between the stress sources. Effort stress for work was significantly greater than for family, finance, and own body. The main effect for group for Effort Stress, $F(2, 58) = 11.55$, $p < .001$, indicated significant differences between the groups. Subsequent post hoc analyses showed C-SADs and S-SADs to rate effort stress significantly higher than controls. The two SAD groups did not differ.

The significant interaction between stress source and group for Effort Stress, $F(5.43, 157.27) = 2.45$, $p = .032$, indicated the groups varied in effort stress between the stress sources. S-SADs and C-SADs rated effort stress for family,

finance and body higher than controls. The groups did not differ significantly for effort for work. Univariate repeated measures analyses for each group revealed significant differences for controls. Controls expended more effort for work than for family, finance, and body. Controls also expended more effort for finance than for family. Neither C-SADs, nor S-SADs, showed significant differences between the stress sources for effort expended.

For Effort Stress the significant interaction between month and stress source, $F(10.17, 621.69) = 3.34, p < .001$, indicated ratings for the stress sources varied across the months. Subsequent univariate repeated measures for each stress source across the months revealed significant differences in effort put up for work. Participants expended more effort for work in March, September, and November, than in January, and July, and more effort in May than in July. Effort put up for family was greater in November than in January, July, and September. Greater effort was expended for body in March than in January, May, and November, and also greater in September than January, and November.

For Part C, polynomial contrasts were conducted for each of the sixteen emotions separately. Univariate $6 \times 4 \times 3$ (Month \times Emotion \times Group) repeated measures ANOVAs were conducted for each category. These were positive somatic emotions (excitation, relaxation, placidity, and provocativeness); negative somatic emotions (boredom, anxiety, anger, and sullenness); positive transactional emotions (virtue, pride, gratitude, and modesty); and negative transactional emotions (humiliation, shame, resentment, and guilt).

Somatic Emotions. Means and standard deviations for eight somatic emotions experienced in the thirty days prior to each bimonthly testing session are presented in two tables. Table 20 contains the four positive somatic emotions, and Table 21 contains the four negative somatic emotions.

Overall individual trend analyses using planned polynomial contrasts on each somatic emotion showed significant quadratic effects for month for relaxation, excitement, placidity, anger, and sullenness; and a significant cubic effect for anxiety. There were no significant effects for provocativeness or boredom. Planned polynomial contrasts on each emotion revealed significant quadratic effects for month for relaxation, $F(1, 58) = 6.27, p = .015$; for excitement, $F(1, 58) = 17.10, p < .001$; for placidity, $F(1, 58) = 4.53, p = .038$; for anger, $F(1, 58) = 4.72, p = .034$; and sullenness, $F(1, 58) = 7.53, p = .008$; and a significant cubic effect for anxiety, $F(1, 58) = 17.52, p < .001$. Figure 19 shows a trough for positive emotions relaxation and excitement, and a peak for placidity, during the autumn/winter months. Figure 19 also shows negative somatic emotions anger, sullenness, and anxiety to show a peak during the autumn/winter months. Anxiety also shows a secondary peak for November.

Overall, for the positive somatic emotions, ANOVA revealed a significant main effect for emotion, and significant two-way interactions between month and emotion and between emotion and group. For the negative somatic emotions, ANOVA revealed significant main effects for month, for emotion, and for group. Significant interactions were also revealed between month and emotion, and between emotion and group.

Table 20

Means and Standard Deviations for Positive Somatic Emotions from the Tension and Effort Stress Inventory Experienced in the Thirty Days Prior to each of Six Bimonthly Testing Sessions by Control, Subsyndromal SAD (S-SAD), and Clinical SAD (C-SAD) Groups

Emotion	Group	January		March		May		July		September		November	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Relaxation	Control	5.00	1.45	4.95	1.29	4.00	1.41	4.68	1.32	4.45	1.44	4.41	1.5
	S-SAD	4.29	1.38	3.48	1.44	3.95	1.36	4.00	1.64	3.95	1.50	4.29	1.76
	C-SAD	4.72	1.41	3.44	1.20	3.78	1.40	4.11	1.28	3.56	1.25	3.89	1.49
Excitement	Control	5.41	0.91	5.14	1.32	4.41	1.84	4.55	1.60	5.14	1.67	5.05	1.43
	S-SAD	4.81	1.50	4.05	1.40	4.14	1.56	4.10	1.22	4.19	1.25	4.48	1.54
	C-SAD	4.56	1.46	4.00	1.41	3.72	1.60	3.50	1.62	4.22	1.59	4.28	1.49
Placidity	Control	3.27	1.28	3.45	1.60	4.05	1.59	3.14	1.36	3.55	1.22	3.09	1.23
	S-SAD	2.95	1.20	3.14	1.46	3.38	1.60	3.24	1.45	3.38	1.56	3.19	1.6
	C-SAD	3.67	1.61	3.50	1.10	3.50	1.54	4.39	1.09	3.11	1.23	3.50	1.62
Provocativeness	Control	2.73	1.70	2.59	1.44	2.77	1.69	2.59	1.37	3.00	1.54	2.82	1.53
	S-SAD	3.71	1.82	3.52	1.75	3.48	1.50	3.57	1.72	3.29	1.45	3.10	1.7
	C-SAD	2.50	1.62	2.67	1.41	3.11	1.75	3.06	1.86	2.67	1.53	2.94	1.7

Note. Ratings for each emotion were made on a 7-point scale (1 = not at all, 7 = very much)

n = 22 Control, 21 S-SAD, and 18 C-SAD participants.

Table 21

Means and Standard Deviations for Negative Somatic Emotions from the Tension and Effort Stress Inventory Experienced in the Thirty Days Prior to each of Six Bimonthly Testing Sessions by Control, Subsyndromal SAD (S-SAD), and Clinical SAD (C-SAD) Groups

Emotion	Group	January		March		May		July		September		November	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Anxiety	Control	2.68	1.49	3.05	1.86	3.55	1.87	3.09	1.80	3.27	2.03	4.09	2.02
	S-SAD	3.71	1.98	4.29	2.03	4.43	1.43	4.00	1.90	4.33	1.56	4.29	1.95
	C-SAD	2.83	1.38	4.56	1.10	4.67	1.50	4.33	1.41	5.11	1.23	4.83	1.69
Boredom	Control	2.68	1.49	2.68	1.86	2.77	1.74	2.91	1.44	2.50	1.57	3.27	1.83
	S-SAD	2.76	1.87	2.62	1.86	2.57	1.43	3.24	1.97	2.62	1.53	2.52	1.75
	C-SAD	2.83	1.89	3.00	2.09	3.56	1.65	3.89	1.64	2.61	1.88	2.78	1.56
Anger	Control	2.00	1.20	2.14	1.28	2.59	1.50	2.59	1.79	2.73	1.61	2.41	1.40
	S-SAD	3.43	1.86	3.95	1.80	3.90	1.58	3.52	2.02	3.57	1.75	3.19	1.57
	C-SAD	2.89	1.28	3.33	1.28	3.67	1.61	3.56	1.89	3.39	1.58	3.89	1.53
Sullenness	Control	2.27	1.42	2.50	1.44	2.23	1.27	2.32	1.43	2.36	1.22	2.27	1.52
	S-SAD	2.57	1.66	2.67	1.68	3.00	1.82	3.10	1.55	2.90	1.55	2.38	1.43
	C-SAD	2.61	1.65	2.39	1.38	3.56	1.42	3.17	1.89	2.06	1.16	2.22	1.40

Note. Ratings for each emotion were made on a 7-point scale (1 = not at all, 7 = very much)

n = 22 Control, 21 S-SAD, and 18 C-SAD participants.

For the positive somatic emotions, the significant interaction between month and emotion, $F(11.36, 658.69) = 2.97, p < .001$, indicated that ratings for the emotions varied across the months. Subsequent univariate repeated measures for each emotion across the months revealed significant effects for relaxation and excitement. Participants were less relaxed in March and May than in January. Participants also showed less excitement in March, May, and July, than in January. Figure 19 shows these differences for relaxation and excitement.

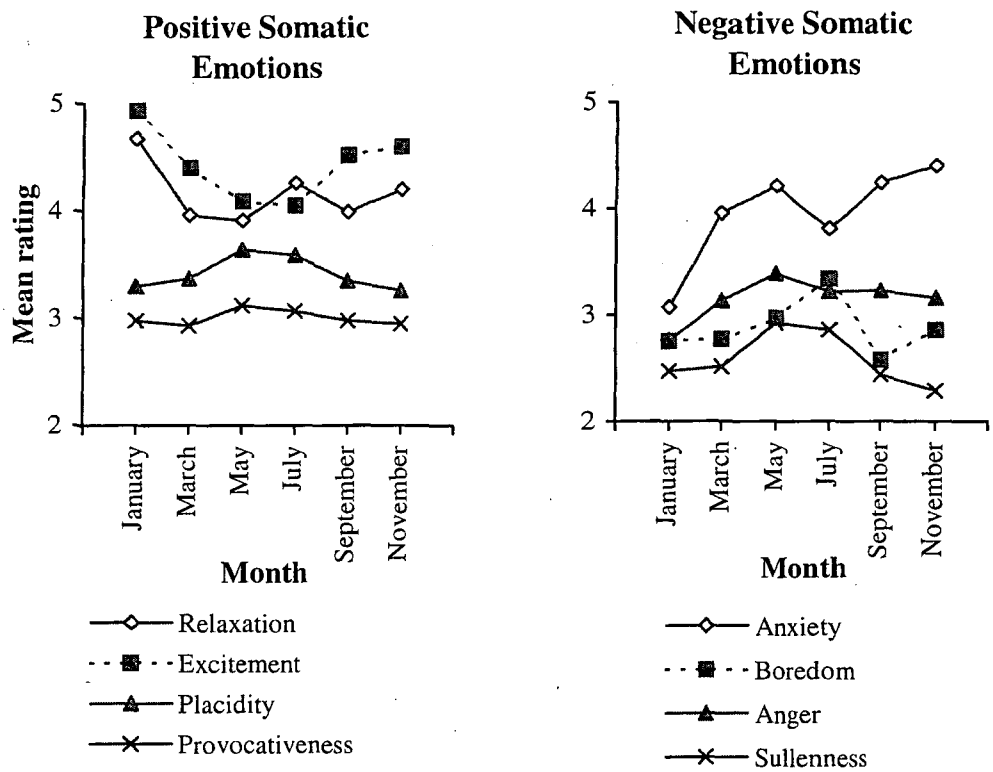


Figure 19. Participant ratings for positive (relaxation, excitement, placidity, and provocativeness) and negative (anxiety, boredom, anger, and sullenness) somatic emotions experienced in the thirty days prior to each of six bimonthly testing sessions

The significant interaction between emotion and group, $F(5.09, 147.71) = 3.53, p = .005$, indicated that the groups varied in their ratings for each emotion. Subsequent univariate analyses between the groups for each emotion revealed significant differences for relaxation and excitement. The two SAD groups were less relaxed and excited than controls, though did not differ from each other.

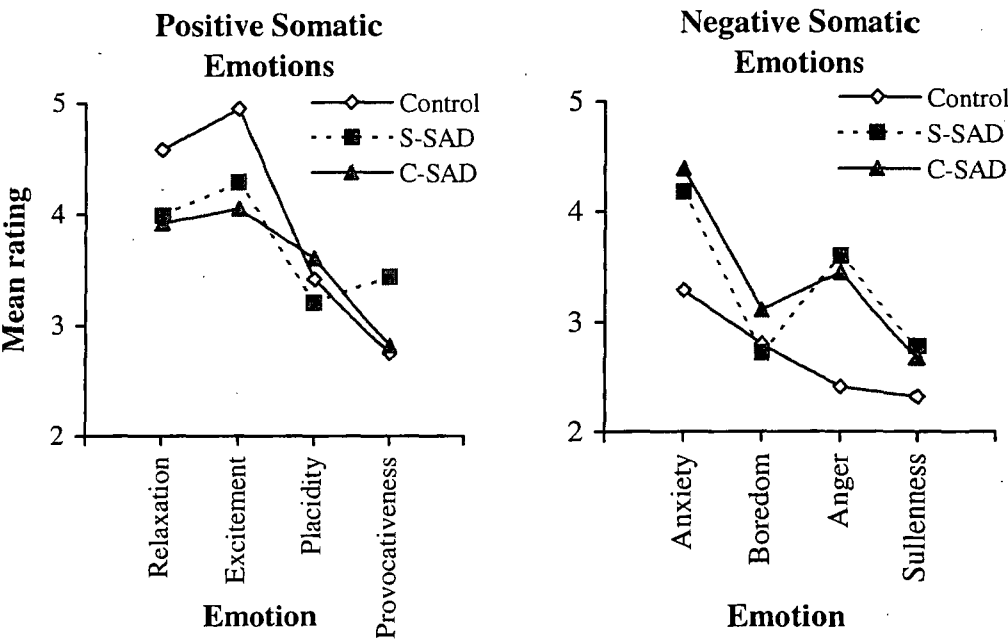


Figure 20. Positive (relaxation, excitement, placidity, and provocativeness) and negative (anxiety, boredom, anger, and sullenness) somatic emotions for Control, Subsyndromal SAD (S-SAD), and Clinical SAD (C-SAD) groups.

For the negative somatic emotions, the main effect for month, $F(4.22, 244.77) = 3.91, p = .004$, indicated the ratings varied across the months. The main effect for group, $F(2, 58) = 3.26, p = .045$, indicated that ratings for negative somatic emotions varied between the groups.

The significant interaction between month and emotion, $F(10.98, 636.61) = 3.81, p < .001$, indicated that the ratings for each emotion varied across the months. Subsequent repeated measures analyses for each emotion across the months revealed significant differences for anxiety and sullenness. Participants showed less anxiety in January than in all other months. Participants are more sullen in May and July than in November.

The significant interaction between emotion and group, $F(5.17, 149.80) = 3.24, p = .008$, indicated that ratings for each emotion varied across the Groups. Subsequent univariate analyses for each emotion between the groups revealed significant differences for anxiety and anger. Figure 20 shows more anxiety and anger for the two SAD groups than for controls.

Transactional Emotions. Means and standard deviations for eight transactional emotions experienced in the thirty days prior to each bimonthly testing session are presented in two tables. Table 22 contains the positive transactional emotions, and Table 23 contains the negative transactional emotions.

Overall, trend analyses using planned polynomial contrasts on individual emotion separately revealed a significant cubic effect for month, and a significant polynomial interaction between month and group, for modesty. Trend analyses also revealed significant quadratic effects for month for humiliation, shame, and guilt. There were no significant effects for pride, gratitude, virtue, or resentment.

Table 22

Means and Standard Deviations for Positive Transactional Emotions from the Tension and Effort Stress Inventory Experienced in the Thirty Days Prior to each of Six Bimonthly Testing Sessions by Control, Subsyndromal SAD (S-SAD), and Clinical SAD (C-SAD) Groups

Emotion	Group	January		March		May		July		September		November	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Pride	Control	3.86	1.81	3.32	1.55	3.32	2.03	3.14	2.12	3.45	1.79	3.64	1.29
	S-SAD	4.05	1.47	3.71	1.38	3.62	1.53	3.67	1.56	3.71	1.42	4.05	1.8
	C-SAD	3.11	1.60	3.17	1.54	3.11	1.71	3.33	1.50	3.06	1.11	3.06	0.94
Modesty	Control	2.95	1.17	3.09	1.72	3.14	1.58	2.68	1.39	3.14	1.81	3.05	1.40
	S-SAD	3.76	1.45	3.24	1.34	3.86	0.91	3.29	1.01	3.19	1.44	3.48	1.40
	C-SAD	2.72	1.36	3.72	1.27	3.50	1.58	2.56	1.38	3.11	1.45	3.28	1.56
Gratitude	Control	4.23	1.85	4.64	1.62	4.45	2.11	4.27	1.75	4.18	1.87	4.55	1.41
	S-SAD	4.43	1.60	4.24	1.51	4.48	1.40	4.62	1.50	3.81	1.50	4.90	1.37
	C-SAD	4.11	1.81	3.78	1.80	4.22	1.52	3.89	1.08	4.17	1.34	4.17	1.29
Virtue	Control	3.27	1.49	3.23	1.54	3.23	1.54	3.27	1.75	3.23	1.38	3.00	1.31
	S-SAD	3.57	1.29	3.05	1.50	3.24	0.94	3.05	1.07	3.57	1.63	3.43	1.63
	C-SAD	2.94	1.16	3.33	1.19	2.67	1.46	2.39	1.20	3.33	1.03	3.06	1.21

Note. Ratings for each emotion were made on a 7-point scale (1 = not at all, 7 = very much)

n = 22 Control, 21 S-SAD, and 18 C-SAD participants.

Table 23

Means and Standard Deviations for Negative Transactional Emotions from the Tension and Effort Stress Inventory Experienced in the Thirty Days Prior to each of Six Bimonthly Testing Sessions by Control, Subsyndromal SAD (S-SAD), and Clinical SAD (C-SAD) Groups

Emotion	Group	January		March		May		July		September		November	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Humiliation	Control	1.62	1.12	1.52	0.81	2.00	1.22	1.90	1.34	1.57	0.93	1.67	1.06
	S-SAD	2.33	1.43	2.57	1.72	2.62	1.69	2.86	1.88	2.38	1.16	2.00	1.14
	C-SAD	2.12	1.45	2.18	1.33	3.18	1.94	1.71	1.21	1.94	1.14	2.24	1.39
Shame	Control	1.52	0.93	1.48	0.98	2.00	1.58	1.62	0.86	1.86	1.28	1.57	0.98
	S-SAD	1.90	1.04	2.19	1.50	2.86	1.46	2.33	1.56	2.14	1.35	2.10	1.14
	C-SAD	2.00	1.37	1.59	0.62	2.71	1.45	2.00	1.17	1.71	0.85	1.94	1.56
Resentment	Control	2.14	1.39	1.95	1.20	2.05	1.40	2.48	1.60	1.95	1.47	1.90	1.37
	S-SAD	2.81	1.63	3.00	1.95	3.14	1.80	2.95	1.86	3.14	1.96	3.05	1.77
	C-SAD	2.76	1.44	2.53	1.37	3.82	1.85	3.00	1.97	2.59	1.37	3.06	2.08
Guilt	Control	1.81	1.08	1.76	1.37	1.95	1.20	1.86	1.01	1.95	1.66	1.81	1.36
	S-SAD	2.52	1.63	2.48	1.63	3.48	1.83	2.90	2.05	2.71	2.00	2.43	1.63
	C-SAD	2.12	1.36	2.18	1.24	3.18	1.70	2.18	1.38	2.18	1.51	2.47	1.59

Note. Ratings for each emotion were made on a 7-point scale (1 = not at all, 7 = very much)

n = 22 Control, 21 S-SAD, and 18 C-SAD participants.

Trend analyses showed a significant cubic main effect for modesty, $F(1,58) = 9.83, p = .003$. A significant interaction between month and group was also shown for modesty, $F(2,58) = 4.08, p = .022$. Subsequent contrasts for each group revealed a significant cubic effect for C-SADs only. Figure 21 shows C-SADs to peak in modesty for March and May. A secondary peak is also evident for November. Trend analyses on individual emotions also showed significant quadratic effects for month for humiliation, $F(1,58) = 7.89, p = .028$, shame, $F(1,57) = 5.21, p = .026$, and guilt, $F(1, 58) = 4.63, p = .036$. Figure 21 shows these emotions to peak during the autumn/winter months.

Overall, for the positive transactional emotions, ANOVA revealed a significant interaction between month and emotion. For the negative transactional emotions, ANOVA revealed significant main effects for month, and for group.

For the positive transactional emotions, the significant interaction between month and emotion, $F(11.37, 659.72) = 2.00, p = .024$, indicated the emotions varied across the months. Subsequent repeated measures analyses for each emotion across the months revealed significant differences for modesty only. Participants showed more modesty in March and May than in July (Figure 21).

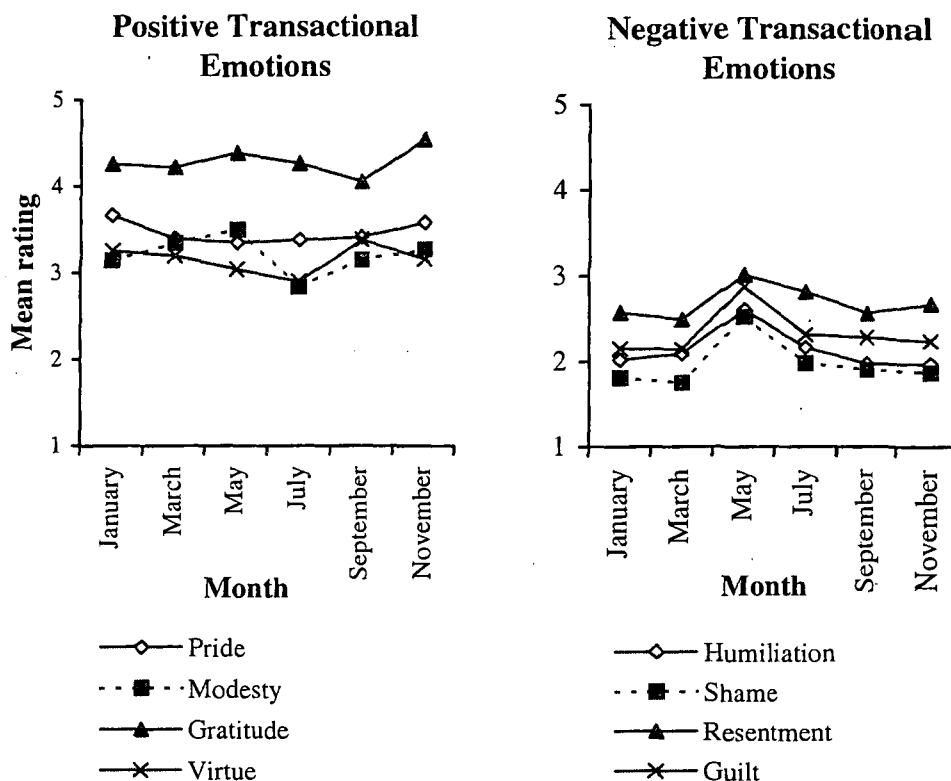


Figure 21. Participant ratings for positive (pride, modesty, gratitude, and virtue) and negative (humiliation, shame, resentment, guilt) transactional emotions experienced in the thirty days prior to each of six bimonthly testing sessions

For the negative transactional emotions, the main effect for month, $F(4.08, 228.39) = 4.44, p = .002$, indicated the ratings varied across the months. Subsequent post hoc pairwise comparisons revealed negative transactional emotions to be rated higher in May, than all other months. The main effect for group, $F(2, 56) = 4.64, p = .014$, indicated that ratings for negative transactional emotions vary between the groups. The S-SAD group rated the emotions significantly higher than controls. The two SAD groups did not differ from each other.

For the current felt stress scale, means and standard deviations are presented in Table 24.

Initial trend analyses using polynomial contrasts were conducted separately for pre-stressor and mid-stressor ratings. For the pre-stressor scale, polynomial contrasts revealed a significant main effect for month, and a significant cubic interaction between month and group. For the mid-stressor scale, polynomial contrasts revealed a quadratic main effect for month.

For the pre-stressor condition, the quadratic main effect for month, $F(1, 58) = 20.64, p < .001$, indicated a seasonal pattern across the months. The cubic interaction between month and group, $F(2, 58) = 4.72, p = .013$, indicated that the seasonal pattern varied across the groups. Subsequent polynomial contrasts for each group separately revealed quadratic main effects for controls, S-SADs, and C-SADs. Figure 22 shows both SAD groups to show a peak for March and May. Controls show a peak in September. A cubic main effect was also revealed for C-SADs, reflecting a secondary peak for September/ November.

For the mid-stressor condition, the quadratic main effect for month, $F(1, 58) = 11.93, p = .001$, indicated a seasonal pattern across the months. Figure 22 shows felt current stress to peak in May at the mid-stressor rating.

Table 24

Means and Standard Deviations for Pre- and Mid-Stressor Task Ratings for the Current Felt Stress Scale from the State Version of the Tension and Effort Stress Inventory by Control, Subsyndromal SAD (S-SAD), and Clinical SAD (C-SAD) Groups at Each of Six Bimonthly Testing Sessions

Time	Group	January		March		May		July		September		November	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Pre-stressor	Control	1.77	1.02	2.05	1.33	2.23	1.54	2.45	1.60	2.64	1.89	1.64	0.79
	S-SAD	2.48	1.36	3.05	1.60	2.95	1.12	2.95	1.28	3.05	1.16	2.19	0.93
	C-SAD	2.36	1.26	3.67	1.57	3.89	1.41	3.56	1.29	3.72	1.78	3.67	1.41
Mid-stressor	Control	2.86	1.73	3.00	1.38	3.00	1.31	3.00	1.41	3.09	1.60	2.86	1.81
	S-SAD	3.48	1.47	3.90	1.41	4.57	1.16	4.10	1.48	4.05	1.12	3.38	1.16
	C-SAD	4.00	1.46	4.11	1.18	4.44	1.38	4.22	1.22	4.33	1.50	4.00	1.57

Note. Ratings were made on a 7-point scale (1 = not at all, 7 = very much)

n = 22 Control, 21 S-SAD, and 18 C-SAD participants.

A repeated measures 6 x 2 x 3 (Month x Time: pre- / mid- Stressor task x Group) ANOVA was also conducted on the current felt stress scale. Overall, ANOVA revealed main effects for month, for time, and for group. ANOVA also revealed a significant three-way interaction between month, time, and group.

The main effect for month, $F(5, 54) = 6.17, p < .001$, indicated current felt stress to vary across the months. The main effect for time, $F(1, 58) = 70.37, p < .001$, showed all participants rated their current felt stress higher mid-stressor task than pre-stressor task. The main effect for group, $F(2, 58) = 8.64, p = .001$, indicated significant differences between the groups.

The significant three-way interaction between month, time, and group, $F(9.49, 275.19) = 1.80, p = .05$, indicated that the groups varied between their pre- and mid- stressor ratings across the months. Post hoc pairwise comparisons showed controls and S-SADs to increase in felt current stress from pre- to mid-stressor in every month. C-SADs increased in felt current stress from pre- to mid-stressor in January and July only. For the pre-stressor condition, C-SADs rated felt current stress significantly greater than controls in March, May, and November. S-SADs also rated felt current stress greater than controls in March and May. A strong tendency in July was due to significantly greater felt current stress for C-SADs as compared to S-SADs and controls. For the mid-stressor condition, the two SAD groups rated felt current stress significantly greater than controls in March, May, July, and September. Figure 22 shows these differences for felt current stress.

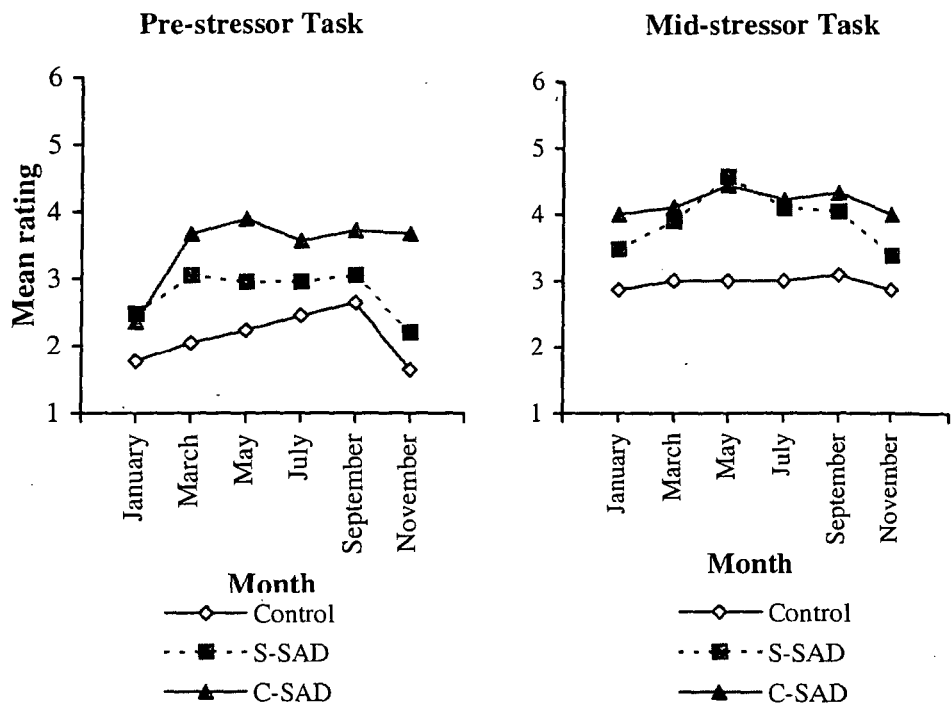


Figure 22. Current Felt Stress ratings at pre- and mid- stressor task for control, subsyndromal SAD (S-SAD), and clinical SAD (C-SAD) groups at six bimonthly testing sessions

The Motivational Style Profile (MSP). Mean self-rated scores and standard deviations for motivational states and tendencies from two testing sessions (winter and summer) are shown in Table 25.

Multivariate 2 x 2 x 3 (Season x Motivational State x Group) repeated measures MANOVAs were used for each pair of motivational states: telic/ paratelic, arousal avoidance/ arousal seeking, defiance/ conformity, as well as each pair of motivational tendencies: optimism/ pessimism, and emotionality/ effortfulness, separately. A multivariate 2 x 2 x 2 x 3 (Season x Mastery/ Sympathy x Autic/ Alloic x Group) repeated measures analysis was used for the four transactional

Table 25

Means and Standard Deviations for Metamotivational States and Tendencies for Control, Subsyndromal SAD (S-SAD), and Clinical SAD (C-SAD) Groups in Winter (July) and Summer (January)

Subscale	January						July					
	Control		S-SAD		C-SAD		Control		S-SAD		C-SAD	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Telic	22.36	4.16	21.05	3.20	22.89	3.65	20.36	4.75	20.55	3.70	21.79	3.15
Paratelic	20.59	2.92	20.23	3.01	18.89	3.57	19.32	3.33	19.23	3.16	18.63	4.03
Arousal-avoiding	20.68	3.05	18.91	2.83	20.63	3.61	19.64	3.23	19.36	3.33	20.42	2.78
Arousal-seeking	19.05	4.15	19.09	4.16	17.00	4.04	17.32	4.19	17.95	3.47	16.58	4.31
Defiant	11.50	3.46	14.14	2.82	12.42	4.88	11.41	4.41	14.36	2.82	12.89	4.48
Compliant	21.64	2.85	19.14	3.31	20.68	3.07	21.23	3.02	20.18	3.22	21.11	3.33
Autic Mastery	19.32	3.41	18.95	3.40	18.63	3.73	18.73	3.94	18.45	2.81	17.95	3.47
Autic Sympathy	20.95	4.37	20.18	4.29	20.42	5.04	19.55	4.15	19.91	4.20	20.47	5.32
Alloic Mastery	23.23	4.05	21.73	4.52	23.42	4.51	22.23	3.77	21.73	4.31	22.05	5.07
Alloic Sympathy	24.73	3.37	23.50	3.43	24.16	2.99	23.73	3.13	23.68	3.14	24.05	3.91
Optimism	22.32	3.90	21.41	3.62	19.63	3.64	21.82	3.95	20.36	3.53	18.11	3.62
Pessimism	10.50	2.77	12.82	4.76	13.95	3.42	10.14	3.64	13.32	4.53	15.11	3.68
Emotionality	17.59	5.07	18.59	4.23	20.16	4.84	16.64	5.15	19.05	4.49	20.42	5.19
Effortfulness	22.77	4.58	20.95	4.60	22.26	3.89	20.91	4.05	20.95	4.15	21.11	4.51

Note. Ratings were made on a 6-point scale (1 = never, 6 = always) and totalled for each subscale.

n = 23 Control, 22 S-SAD, and 18 C-SAD participants.

variables: autic mastery, autic sympathy, alloic mastery, and alloic mastery.

Overall, the MSP revealed significant main effects for all pairs of motivational states and tendencies. Main effects for Season were also revealed for the telic, arousal, emotionality, and transactional pair analyses. There were no main effects for group. The MSP also revealed several two-way interactions. For the optimism/ pessimism pair of subscales, interactions for Motivational Tendency x Group, and Motivational Tendency x Season were revealed. For the defiance/ conformity pair of subscales, a Motivational state x Group interaction was revealed. For emotionality/ effortfulness, a significant Season x Group interaction was revealed.

For the telic/ paratelic pair of subscales, the main effect for motivational state, Pillai's Trace = .173, $F(1, 60) = 12.51$, $p = .001$, indicated a significant difference between the two states. Participants were more telic than paratelic. The main effect for season, Pillai's Trace = .201, $F(1, 60) = 15.13$, $p < .001$, indicated a significant difference between the two seasons. Higher scores were recorded for summer as compared to winter.

For the arousal avoidance/ arousal seeking pair of subscales, the main effect for motivational state, Pillai's Trace = .152, $F(1, 60) = 10.73$, $p = .002$, indicating a significant difference between the states. Participants showed significantly more arousal avoidance than arousal seeking. The main effect for season, Pillai's Trace = .135, $F(1, 60) = 9.37$, $p = .003$, indicated a significant difference between the seasons. Participants reported higher scores in summer as compared to winter.

For the defiance/ conformity pair of subscales, the main effect for motivational state, Pillai's Trace = .702, $F(1, 60) = 141.10, p < .001$. The significant interaction between motivational state and group, Pillai's Trace = .124, $F(2, 60) = 4.24, p = .019$, indicated that the groups vary differentially between the states. Figure 23 shows these group differences. All groups were more compliant than defiant. The S-SAD group is more defiant than controls.

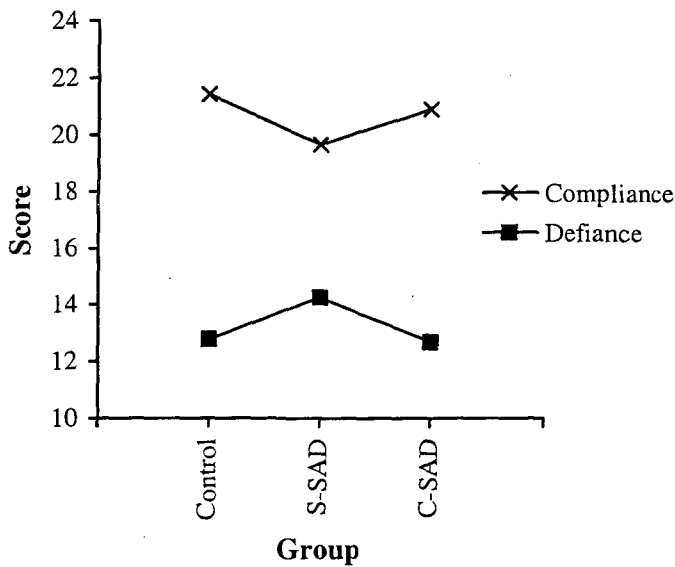


Figure 23. Compliance and defiance scores from the Motivational State Profile for control, subsyndromal SAD (S-SAD), and clinical SAD (C-SAD) groups

For the transactional variables, the main effect for mastery/ sympathy, Pillai's Trace = .279, $F(1, 60) = 23.20, p < .001$, and the main effect for autistic/ alloic, Pillai's Trace = .570, $F(1, 60) = 79.67, p < .001$, indicated significant differences between each pair of states. Overall, participants showed more sympathy than mastery, and were more alloic than autistic. The main effect for season for the

transactional variables, Pillai's Trace = .077, $F(1, 60) = 5.03$, $p = .029$, indicated a significant difference between the seasons. Participants recorded higher scores for the transactional variables in summer than in winter.

The main effects for motivational tendency for the emotionality/ effortfulness pair of subscales, Pillai's Trace = .174, $F(1, 60) = 12.63$, $p = .001$, indicated a significant difference between the tendencies. The main effects for season for emotionality/ effortfulness, Pillai's Trace = .07, $F(1, 60) = 4.55$, $p = .037$, indicated a significant difference between the seasons. The significant interaction between season and group, Pillai's Trace = .109, $F(2, 60) = 3.65$, $p = .032$, indicated that the groups vary differentially between the seasons. Subsequent post hoc comparisons between winter and summer for each group showed significant differences for controls only. Controls showed a greater motivational tendency for emotionality/ effortfulness in summer than in winter (see Figure 24). There were no differences between the groups either in summer or winter.

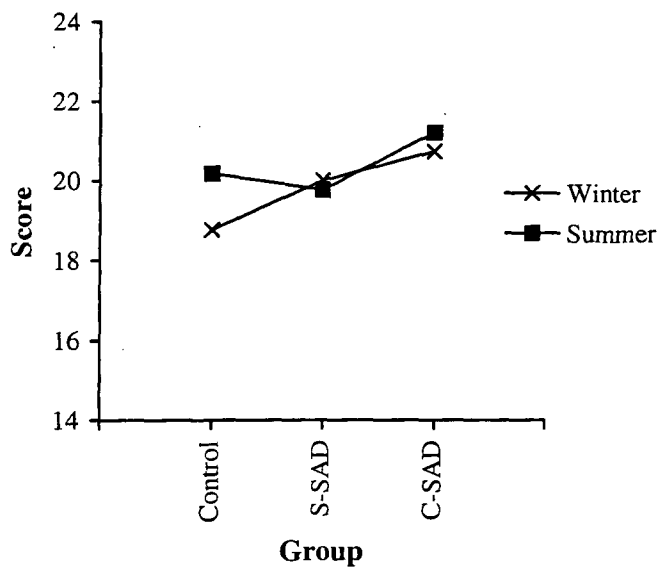


Figure 24. Emotionality/ effortfulness subscale scores from the Motivational State Profile for control, subsyndromal SAD (S-SAD), and clinical SAD (C-SAD) groups

The main effects for motivational tendency for optimism/ pessimism, Pillai's Trace = .647, $F(1, 60) = 110.04$, $p = .001$, indicated a significant difference between the tendencies. For optimism/ pessimism, the significant interaction between motivational tendency and group, Pillai's Trace = .206, $F(2,60) = 7.78$, $p = .001$, indicated the groups varied significantly across seasons. All groups were significantly more optimistic than pessimistic. C-SADs showed less optimism than controls. Both SAD groups showed more pessimism than controls.

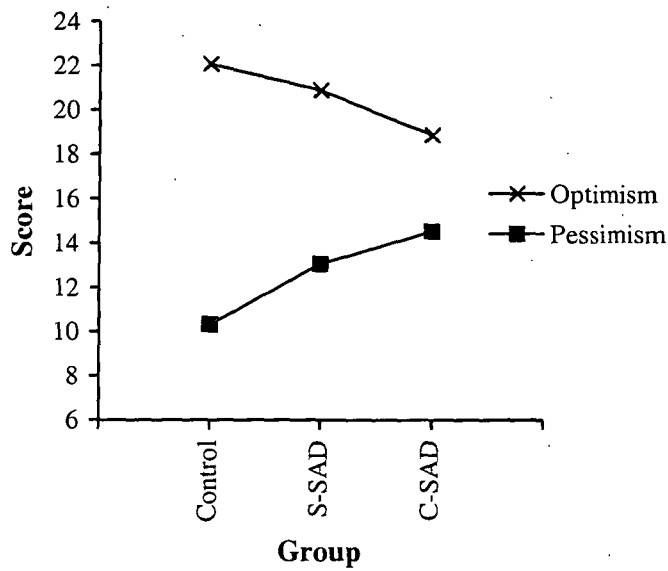


Figure 25. Scores for optimism and pessimism tendencies from the Motivational State Profile for control, subsyndromal SAD (S-SAD), and clinical SAD (C-SAD) groups

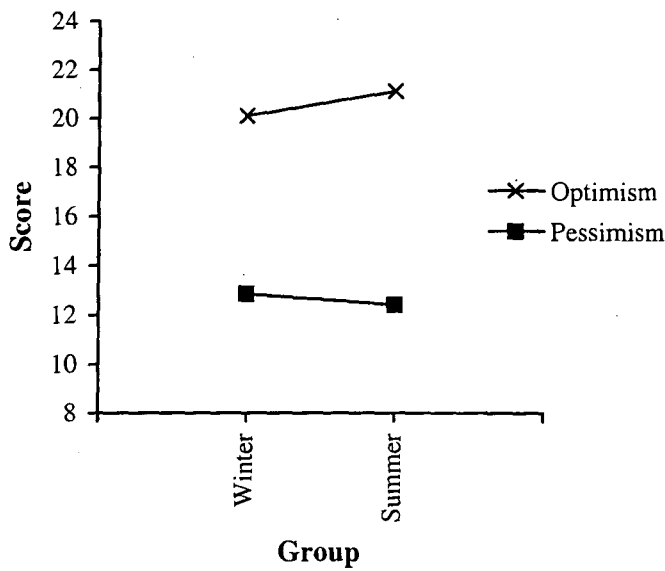


Figure 26. Scores for optimism and pessimism tendencies from the Motivational State Profile in summer and winter

The significant interaction for optimism/ pessimism between motivational tendency and season, Pillai's Trace = .074, $F(1, 60) = 4.79$, $p = .032$, indicated that participants vary differentially in optimism and pessimism score between winter and summer. Subsequent post hoc analyses showed optimism to be significantly greater than pessimism in both seasons. Participants were more optimistic in summer than in winter, though did not differ significantly between the seasons for pessimism.

Summary of results for emotion analyses. Several main results were shown from the questionnaires (TESI and MSP) for experience of emotion and arousal. The SAD groups felt more tension stress, and expended more effort, for family, finance, and body, than controls. The groups did not differ in either felt tension stress, or effort expended for work. However, for all participants tension stress felt and effort expended for work was increased for March/ May, and September/ November.

The SAD groups felt less relaxation and excitement, and more anxiety and anger than controls. Regardless of emotion, SADs rated the negative transactional emotions greater than controls.

Several positive emotions were decreased, and negative emotions increased during the autumn/ winter months. Participants overall showed *less* relaxation, excitement, and placidity, and *more* anger, sullenness, humiliation, shame, and guilt, during the autumn winter months.

The felt current stress scale was greater at the mid-stressor task rating than at the pre-stressor rating, and greater for the SAD groups in autumn/ winter.

For the MSP, the following main findings were shown. Overall all groups were more compliant than defiant, however the S-SAD group was more defiant than controls. For the emotionality/ effortfulness motivational tendencies, the SAD groups did not differ between summer and winter, however controls scored higher in summer than in winter. The motivational tendency scales for optimism and pessimism interacted with both group, and season. All groups were more optimistic than pessimistic, though the SAD groups were more pessimistic than controls. All participants were more optimistic in summer than in winter, though pessimism did not differ between summer and winter. Regardless of group, all subscales showed more extreme measures for summer than winter.

Discussion

Screening questionnaire

The SPAQ differentiated between the groups for all symptoms, and for the GSS and GR. The SAD groups rated a greater degree of change to all symptoms as compared to controls. C-SADs also rated these seasonal changes greater than S-SADs for all symptoms except sleep. The SAD groups reported a greater degree of weight fluctuation (WtFluct) across a twelve-month period as compared to controls, though did not differ from each other. SADs slept more hours in winter than for any other season, and also slept more in autumn as compared to summer. Controls showed no difference in the number of hours slept between the seasons.

Depression and behavioural symptoms

For longitudinal study 1, the two SAD groups showed predicted increases in depression ratings during winter as indexed by the Beck Depression Inventory with addendum. The two SAD groups also showed the expected increases in behavioural symptoms during winter as indexed by the WMI scale for atypical vegetative symptoms (AVS). The WMI scales for depressed mood and ideation (DMI) and for hypomania mood and ideation (HMI) showed main effects for month, and for group. Overall, DMI was increased and HMI decreased for winter. The two SAD groups showed more extreme scores for the DMI and HMI scales than controls. For the remaining WMI scale for typical vegetative symptoms (TVS), random episodes of depression shown across the year were not indicative of SAD.

Group differences were revealed for two of the sleep quality scales; good/ bad nights and restful/ disturbed nights. The two SAD groups had worse nights and more disturbed nights as compared to controls. The two SAD groups did not differ from each other. The study showed changes to food preference across the months. C-SADs and S-SADs were more likely to prefer carbohydrates and fats during the autumn/ winter months as compared to controls. This trend was not evident during the spring and summer.

Constitutional measures

BMI varied across the months, though percentage bodyfat and fitness level did not. A higher BMI was shown for winter as compared to autumn and spring reflecting the SAD symptom of overeating and weight gain in winter, and the subsequent remission in spring. BMI was also higher in January than in March, possibly reflecting increased consumption over the Christmas and holiday period.

On the other hand, no differences were shown for percentage body fat or fitness level either between the groups or across the months. Percentage bodyfat was calculated from the sum of biceps, triceps, suprailiac, and sub-scapula. It is possible that these sites do not reflect fat deposited from increased winter eating.

Psychophysiological measures

The psychophysiological measures for heart rate show initial support for hypo-arousal or an increased vagal tone during winter. Regardless of group, baseline heart rate was lower during the autumn months as compared to November. On the other hand, the expected increase in RSA for the SAD groups in winter was not found for this study. There were no month or group differences for either actual RSA or RSA difference score. These findings show some support for Austen and Wilson's (2001) pilot study of an *increase* in vagal tone. The finding was indicative of increases in sleep, suggesting similarities with hibernation. On the other hand, previous findings (Rechlin et al., 1994; Rechlin et al., 1995) have shown a *decrease* in vagal tone for NSD, supporting a distinction between SAD and NSD. Decreases in RSA are typical in negative emotion, while increases in RSA are characteristic of increases in sleep (Porges, 1995).

In contrast to the hypo-arousal or increased parasympathetic activity from vagal tone, SCL was increased for both baseline and stressor conditions for C-SADs during autumn suggesting high physiological arousal, due to increased sympathetic arousal for C-SADs. In addition to autumn increases for SCL, C-SADs also showed increased SCL at both baseline and stressor levels during November as compared to control participants, most likely representing increased physiological arousal during the university end of year examination period.

Regardless of group, all participants decreased in RSA and increased in HR and SCL from baseline to stressor conditions as predicted. For the C-SAD group, the increase in HR from baseline to stressor condition was greater than for S-SADs and controls. The expected vasoconstriction for pulse height was not shown. Overall, pulse height increased between baseline and stressor, a vasodilation indicating the withdrawal of sympathetic activity. Two possibilities may account for this unexpected finding for pulse height. Firstly, baseline measures of pulse height may have been constrained due to initial anxiety in a laboratory setting, which dissipated over the session and thus produced dilation at the stressor level. Alternatively, the dilation may have been due to the cold environmental conditions. Even though the laboratory temperature was controlled year-round to 22° C. According to the Law of Initial Values, Lovallo and Zeiner (1975) showed vasodilation for participants after a cold pressor stressor test under a room temperature of 12° C. In contrast participants given the cold pressor stressor test under room temperature conditions of 22° C and 32°C showed the expected vasoconstriction. Both explanations for the unexpected vasodilation or withdrawal of sympathetic arousal suggest the importance of control measures for pulse height at baseline. Face skin temperature increased from baseline to stressor conditions for all participants, which parallels the increase in finger pulse height. In contrast, finger skin temperature showed an increase from baseline to stressor levels in November only, and is consistent with the November increase in SCL.

All differences between baseline and stressor relate to changes in physiological functioning due to the stress of the additional cognitive load. The

mental arithmetic stressor task showed no group differences. On the other hand, the current felt stress scale, given to validate the stressful nature of the mental arithmetic task, differentiated between the groups and across the months. S-SADs and controls consistently increased from baseline to stressor levels confirming the stressful nature of the mental arithmetic stressor task. In contrast, C-SADs showed an increase in current felt stress from baseline to stressor levels for January and July only. Two possibilities could explain this finding. Firstly, C-SADs had high felt stress at pre-stressor stage for March/ May and September/ November as compared to controls. The C-SAD group have experienced high levels of stress at the commencement of the experimental procedure, and not increased any further during the mental arithmetic task.

Emotion

Tension stress felt, as well as effort put up, for the *work* stress source were increased for the months of March, May, September, and November. This result most likely relates to the university semester months, where greater levels of effort are required to cope with a high workload. The remaining two months, July and January were holiday periods. The two SAD groups felt more tension stress from the remaining three stress sources; family, finance, and own body, and also expended a greater effort to cope, as compared to controls.

For both the positive and negative somatic emotions, emotion interacted with month. For the positive emotions, regardless of group participants were less relaxed and excited, but more placid in autumn/ winter. For the negative somatic emotions, participants showed more anxiety and sullenness during autumn and winter. Placidity and sullenness represent low arousal, while anxiety represents high

arousal. These emotions suggest conflicting felt arousal, and support the differences shown in the psychophysiology measures. For example, felt emotions such as placidity and sullenness are indicative of low levels of arousal and supportive of an increased vagal tone, thus implicating a state of torpor or hibernation. In contrast anxiety represents a high felt physiological arousal and may explain the increase in SCL. Emotion also interacted with group in both positive and negative somatic emotion categories. Regardless of month, the two SAD groups felt less relaxed and excited, and also felt more anxiety and anger than controls.

While somatic or felt arousal may assist in understanding physiological arousal, the transactional emotions assist in understanding behaviour. All participants were shown to experience more negative transactional emotions during May than any other month. Overall, regardless of month S-SADs experienced more negative transactional emotion than controls.

Motivational dominance

The MSP showed several findings of interest. Overall, participants were more telic, arousal avoiding, and compliant than the corresponding states: paratelic, arousal seeking, and defiance respectively. Participants also showed more sympathy than mastery, and were more alloic than autic. Whilst participants overall were more compliant than defiant, the S-SAD group were more defiant than control participants. The groups were also differentiated on the motivational tendency pair of subscales: optimism/ pessimism. Overall all groups were more optimistic than pessimistic. C-SADs were less optimistic than controls, while both SAD groups were more pessimistic than controls. Seasonal differences were evident for several scales from the MSP indicating more extreme scores for summer as compared to

winter, and will be described more fully in the following section on seasonality in the general population.

Seasonality in the general population

Several variables were shown to vary with the seasons for control participants. Seasonality was shown for all participants for two WMI scales. These scales were for depressed mood and ideation (DMI) (e.g., 'I have felt guilty', 'I have felt that life is not worth living') and for hypomania mood and ideation (HMI) (e.g., 'I have been efficient at work', 'I have felt happy or elated'). Regardless of group all participants showed higher DMI scores, and lower HMI scores for May. The sleep quality scale, refreshed/ fatigued showed seasonality. Regardless of group, participants were more fatigued for the autumn and winter months, than for spring and summer.

Seasonal variation was shown for baseline predicted RSA, with higher scores for March, May, July, and September as compared to January. Baseline HR varied with the seasons showing a trough during autumn as compared to November. Control participants were increased in SCL for May and July as compared to all other months, further supporting the pilot study (Austen & Wilson, 2001) as well as previous findings (Neumann, 1968; Venables & Christie, 1973). Lower finger and face temperatures were recorded during July as compared to the spring and summer months, consistent with Gardner-Medwin et al.'s (2001) previous finding.

Tension stress felt from work, as well as effort expended to cope showed seasonality, being increased during the university semester months. Seasonality was shown in several emotions from the TESI. All participants felt less relaxed, less

excitement, and more placidity during the autumn/ winter months. Participants also felt more anger, sullenness, humiliation, shame, and guilt during autumn/ winter months.

The MSP, administered twice, in January and July showed seasonal differences with more extreme scores for summer as compared to winter. All participants showed higher scores for summer as compared to winter for the telic/ paratelic scale, the arousal avoidance/ arousal seeking scale, and the combined transactional scale. For the emotionality/ effortfulness scale, controls scored higher in summer as compared to winter, though the SAD groups did not differ between the two seasons. All participants were more optimistic in summer as compared to winter, though pessimism did not differ between the seasons.

Implications

The symptoms of SAD were shown in the SAD participants for autumn/ winter with remissions during spring/ summer. More importantly the specificity to season of the decreases to mood, along with the associated atypical vegetative behavioural symptoms gives a clear-cut distinction from NSD. The SAD groups rated depression and associated atypical symptoms significantly greater than controls in the autumn and winter months. The C-SAD group also scored lower for hypomania mood and ideation than controls in May and July, while the S-SAD group were lower in July. Additional evidence supporting the SAD symptoms was also shown. For example, a lower level of sleep quality was shown for SADs as compared to controls. Further, greater percentages of the two SAD groups showed a preference for eating fats and carbohydrates during the autumn/ winter months as compared to controls, with greater percentages of C-SADs than S-SADs. This

preference for fats and carbohydrates was not evident for spring/ summer. Further, preference for protein did not differ either across the twelve-month period or between the groups.

Physiological differences have also been shown to differ between SAD and NSD. In a pilot study that was a forerunner to Longitudinal Study 1, Austen and Wilson (2001) showed an increased vagal tone during winter as indexed by an increase in RSA together with a decrease in HR, indicative of hypo-arousal and suggesting similarities with a state of torpor or hibernation. Longitudinal Study 1 sought to determine autonomic arousal in SAD and to extend previous findings. Increases to actual RSA were not shown in the current study. However as predicted RSA has changed the underlying dynamics have altered. It is possible that the RSA system has maintained a consistent RSA output by accommodation to changes in some of the operating dynamics of that system. Parasympathetic activity is likely to be involved in the decrease in HR for winter indicating initial support for an increase in vagal tone. Additional support for hypo-arousal was shown from felt arousal/ emotion in the study with participants showing greater placidity as well as sullenness during autumn/ winter. These findings differ from NSD where a decreased vagal tone or hyper-arousal has been shown (Rechlin et al., 1994; Rechlin et al., 1995). Increases to SCL were shown, perhaps indicative of increased levels of anxiety for the SAD groups. Anxiety is characteristic in NSD. Thus it is likely that both branches of the ANS are implicated. An increase in parasympathetic tone may have produced the decrease in HR, while an increase in sympathetic arousal would have resulted in the increase in SCL.

The study also sought evidence for SAD being an extension of seasonality in the general population. The groups were differentiated for depression and all behavioural symptom scales on the screening questionnaire for Longitudinal Study 1 participant selection, except sleep. Thus, the mood and behavioural symptoms that validated group selection may initially suggest seasonality to be on a continuum. However Chapter 4 noted that any apparent continuum possibly relates to the lineal nature of the global seasonality score (GSS) range on the screening questionnaire, SPAQ. Any continuum in seasonality would require more extreme measures in variables for the C-SADs as compared to controls, with S-SADs showing measures in between C-SADs and controls. On the other hand, the sleep scales do not differentiate between the two SAD groups. Both SAD groups reported greater seasonal changes to sleep on the SPAQ, though they did not differ from each other. Two of the sleep quality scales also showed the SAD groups to differ from controls; though not from each other. SADs rated their sleep quality worse, and reported more disturbed nights than controls. Regardless of group, all participants were more fatigued in autumn/ winter.

Further, the psychophysiological measures are not indicative of a continuum, but may in fact suggest differing underlying mechanisms between subsyndromal and clinical levels of SAD. Group differences were not shown for actual RSA, RSA difference scores or pulse height percentage. For HR, the C-SAD group showed a greater response to the mental arithmetic stressor task, while this difference does not vary between controls and S-SADs. For SCL, controls showed a peak for May and July and C-SADs showed a peak in the autumn months reflecting increases to arousal. In contrast, S-SADs did not show this increased arousal.

The trait version of the TESI has shown several differences for the SAD groups overall, regardless of month. For example the SAD groups felt less relaxed and excited, and felt more anxiety and anger, as compared to controls. The S-SAD group felt more negative emotions than controls. Several findings from the MSP add further support to the suggestion that the SAD groups show consistent motivation style differences from controls. For example, the SAD groups did not differ between summer and winter on the scale for emotionality/ effortfulness. In contrast, controls increased on this scale for summer as compared to winter. Participants did not differ in pessimism between summer and winter, though were less optimistic in winter as compared to summer. However, the groups were differentiated on the pessimism scale, with the SAD groups being more pessimistic than controls. From the motivational styles, whilst all groups were more compliant than defiant overall, the S-SAD group was more defiant than controls.

Several theories explaining the pathophysiology underlying SAD were outlined in Chapter 6. For example, early theories involved melatonin, circadian rhythm disruption, and abnormalities to neurotransmitters. More recent theories suggest that a combination of pathophysiologies may be the key to understanding SAD and include the sympatho-adrenal system. Several researchers show support for hypo-arousal (e.g., Lam & Levitan, 2000; Putilov & Danilenko, 1998) that shows similarities with hibernation and provides a distinction from NSD. In addition, support could be shown for the dual vulnerability hypothesis (Young et al., 1991) that states that all SAD sufferers experience initial seasonal disturbances to sleep and energy, followed by depression in those who are vulnerable. Longitudinal Study 1 has not shown differences between the SAD groups for sleep and offers support to a metabolic component of SAD being an extension of the

seasonal change to mood and associated behavioural symptoms seen in the general population. More extreme measures for the C-SAD group from the current study (e.g., greater depression, greater tension stress felt, anxiety, guilt, and pessimism) suggest the vulnerability of the C-SAD group to additional depression.

The study has also shown the symptoms to some degree in control participants, supporting findings from previous literature (e.g., Kasper et al., 1989). All participants showed depressed mood and ideation levels during winter that was greater than spring/ summer levels. All participants also showed lower levels of hypomania mood and ideation during winter as compared to spring/ summer. All participants were more fatigued during autumn/ winter as compared to spring/ summer. Emotion, or experience of arousal varied with the seasons. Regardless of group, participants felt less relaxed and excited, but more anger, sullenness, humiliation, shame and guilt during autumn/ winter as compared to spring/ summer. Further, all participants were more optimistic in summer than in winter.

Future directions

Longitudinal Study 1 has shown evidence for hypo-arousal underlying the seasonal component of SAD, thus implicating a state of torpor and showing similarities with hibernation. Confirmation of an increase in vagal tone is required. Further, energy levels have not been determined across the twelve-month period. However the finding from Chapter 4 indicating the importance of metabolic factors in SAD, together with similarities between SAD and hibernation, and the involvement of the autonomic nervous system suggest that a more extensive investigation of energy levels is warranted. Further examination of the nature of the additional sleep in SAD is also warranted. Further investigation of metabolism in

SAD may also assist in understanding any distinction between clinical and subsyndromal levels of SAD.

Predicted RSA was calculated to enable the determination of any cognitive load component independent of respiratory changes during the experimental procedure. Actual RSA and difference RSA (the difference between actual and predicted RSA) showed no seasonal pattern. In contrast, seasonality was shown for the baseline level only of predicted RSA with a peak for March, May, July, and September as compared to January. To locate the source of seasonality for baseline predicted RSA, further analyses were conducted using the slope and intercept as substituted into linear regression equations. However, analyses still showed no differences. Seasonality, or circannual rhythms have not previously been shown in predicted RSA, thus the finding may have important implications for future testing procedures where comparisons across time are required. Hence, further research is warranted to determine any underlying mechanism creating the seasonality in predicted RSA.

A tendency for more extreme scores was shown on the MSP in summer as compared to winter. Control participants appear to adjust to cope with any changes to energy levels, mood and/ or fatigue during the autumn/ winter months. On the other hand, the SAD groups showed greater levels of pessimism that did not vary between summer and winter, thus indicating a tendency to remain in a negative state regardless of season. Apter & Desselles (2001) suggest that individual states change over time according to their attitudes, circumstances, and situations. The finding suggests that further research across a twelve-month period is warranted, firstly to

document changes to motivational styles and tendencies in control participants and seasonally, to understand the negativity shown in SAD participants.

Conclusions

The specificity to the autumn/ winter months of mood and the atypical vegetative behavioural symptoms showed a clear-cut distinction from non-seasonal major depressive disorder. Additional physiological findings from the study show some support for differential underlying mechanisms between SAD and NSD, with additional future research recommended for further confirmation.

SAD has generally been seen at the extreme end of a continuum of seasonal change/ disturbance to mood and atypical vegetative symptoms ranging from those who experience little or no seasonal change, through mild to moderate symptoms, to those who experience SAD with severe disruption to their everyday functioning during the autumn/ winter months. However, evidence from Longitudinal Study 1 is consistent with dual mechanisms operating in SAD. Many differences are shown between the two SAD groups and the control group, but in many instances the two SAD groups do not differ from each other. In contrast, measures where C-SADs do show a more extreme value than S-SAD, for example, depression, preference for fats and carbohydrates, stress, anxiety, and pessimism, are indicative of trait measures or underlying characteristic tendencies. The study produced findings consistent with Young et al.'s (1991) dual vulnerability hypothesis that proposes that all SAD sufferers experience seasonal disturbances to sleep and energy levels, followed by depression in vulnerable individuals. For the C-SAD group, seasonal disturbances may be compounded by abnormalities to both branches of the ANS. Firstly, a seasonal component from an increase in parasympathetic tone as indexed

by vagal tone and felt emotions such as placidity, sullenness suggesting hypo-arousal and showing similarities with hibernation. Secondly, a depression component from an increase in sympathetic arousal as indexed by increases in SCL and showing underlying trait characteristics of vulnerability to depression along with increases in stress and anxiety.

Chapter 8

Longitudinal Study 2 - Cognitive Performance

Chapter 8: Longitudinal Study 2: Cognitive Performance

“..... cold weakens the mental activities.”

Galen, trans., 1976, p. 81.

Cognitive impairment has been shown in non-seasonal depression (NSD) (e.g., Miller, 1975; Raskin, Friedman, & DiMascio, 1982). For example, deficits have been shown to Digit Span forward and backward (Breslow, Kocsis, & Belkin, 1980), psychomotor speed (Weckowicz, Tam, Mason, & Bay, 1978), concentration, abstract thinking, and accuracy (Raskin et al.). The nature of these deficits to cognitive performance in NSD is unclear, and may be either impairments to the neuro-physiology, or resulting from depressed ideation characteristics, for example, lack of motivation, and inability to concentrate.

Whilst the thesis suggests differing underlying physiological mechanisms between SAD and NSD (refer chapter 5), the winter symptoms of SAD include inability to concentrate, lack of energy, and fatigue. Further, depressed ideation in SAD includes negative thinking pattern, low self-esteem, and lack of motivation (Rosenthal, 1993). Chapter 6 notes the desynchronisation of circadian rhythms in SAD. Healy (1987) has suggested that this disruption to circadian rhythms may cause impairments to memory, concentration, and learning. Hence it seems possible that impairments to cognitive efficiency may also present in seasonal depression.

Cognitive performance in SAD

Several studies have been conducted to determine cognitive efficiency in SAD during winter, firstly to determine any deficits to cognitive functioning (e.g., attention, memory, psychomotor speed, spatial functions), and secondly to assist in understanding any underlying pathophysiology in SAD.

Drake, Schwartz, Turner, & Rosenthal (1996) found both SAD and control participants showed impaired performance on a Stroop task in winter as compared to summer. Spinks and Dalgleish (2001) also found SAD participants to respond more slowly to an emotional Stroop task in winter than in summer. Further, the participants were slower in responding to negative and seasonal relevant words than to neutral words and zeros. However these findings for the Stroop task may require confirmation as (a) for both studies experimentation was conducted in winter first, then the following summer suggesting that a practice effect can not be ruled out, and (b) no control group was used for the Spinks and Dalgleish study. Others (e.g., Austen & Wilson, 1998; Michalon et al., 1997) showed no differences between SAD and control groups in a Stroop task either in winter, or in a non-winter period.

Mixed findings have been shown for memory. For example, Austen and Wilson (1998) showed deficits to short term memory as indexed by the Digit Span forward condition in subsyndromal SAD during winter as compared to controls. In contrast, Michalon et al. (1997) showed no differences to verbal memory or attention on several tasks from the Wechsler Memory Scale.

SAD sufferers may have impairment to their ability to process visuo-spatial information. Several studies have shown impairments in winter that do not remit for summer. O'Brien et al. (1993) showed SAD patients to be slower on a pattern recognition task and a spatial recognition task as compared to controls. On remission from depression, performance to pattern recognition improved, but spatial recognition remained impaired as compared to controls. Similarly, Michalon et al. (1997) found deficits to visual memory and visual construction tasks in winter that did not remit for summer. Additional light is important in the remission of symptoms (Chapter 5). Hence any impairments in the visual pathway, either showing a return to normal with therapy, or remaining abnormal, may assist in further understanding vulnerability and pathophysiology underlying SAD.

Hemispheric specialisation

The right visual field or left hemisphere is generally thought to be involved in processing of verbal information. In contrast, the left visual field, or right hemisphere, has been linked to spatial processing (Levy, 1969; Springer & Deutsch, 1985). The processing of negative emotion is also thought to be in the right hemisphere (e.g., Porges, 1995; Tucker, 1981). A review by Nasrallah (1982) shows an association between affective disorders and right hemisphere dysfunction, however methodological and diagnostic limitations make any overall interpretation difficult. Hemispheric asymmetries have been shown in EEG studies in SAD. Allen, Iacono, Depue, and Arbisi (1993) found greater *left*

frontal alpha activity in SAD during winter before and after light therapy, as compared to right frontal and controls. On the other hand, greater *right* parietal alpha activity was shown before light therapy, but returning to normal after light therapy. Allen et al.'s finding dissociates between the frontal and parietal findings. The left frontal alpha finding represents a trait measure specific to SAD and may have implications for vulnerability to SAD. In contrast, the right parietal finding has been shown previously in NSD (Davidson, Schaffer, & Saron, 1985) and may be state dependent in depression. Using a PET scan procedure, Cohen et al. (1992) showed lower glucose metabolic rates in the right anterior frontal region for SAD participants. This lowered metabolic rate was shown to have a strong relationship to the atypical symptoms in SAD and is consistent with catecholaminergic and serotonergic abnormalities in SAD.

Using a dual task paradigm with a left/ right hand tapping task concurrently with a verbal task, Volf, Senkova, Danilenko, and Putilov (1993) showed lateralisation of language function in SAD participants using a pre- and post- light therapy experiment. Their findings were consistent with a shift of laterality in verbal function from the left to the right hemispheres in SAD during winter as compared to controls implying that SADs have a tendency to process verbal tasks with the right hemisphere. After treatment with light therapy the abnormalities disappeared and there were no differences with controls.

Seasonal variation in the general population

Seasonal variation to mood and atypical behavioural symptoms is evident in the general population during winter (e.g., Lacoste & Wirz-Justice, 1989; Terman, 1989). Evidence for fatigue, low energy levels, lack of motivation, and inability to concentrate may implicate problems in winter in control participants. The suggestion has also been made that subtle deficits to performance may also be evident during winter in the general population (Lacoste & Wirz-Justice).

Hemispheric dominance has also been shown to shift with the seasons in control participants. Corbera (1995) conducted a Posner-type hemispheric asymmetry task with verbal and spatial stimuli. Results showed the expected specialisation pattern of increased performance in the left hemisphere for verbal tasks, and in the right hemisphere for spatial tasks. The results also showed increased performance for the left hemisphere in spring, and the right hemisphere in autumn. Corbera's findings of an autumn shift of laterality to the right hemisphere in controls may be due to the tendency for decreases in mood in autumn in the general population. The finding may also be of assistance in understanding any lateralization in SAD.

The current longitudinal study was undertaken to investigate cognitive efficiency in seasonal affective disorder at clinical and subsyndromal levels, and in control participants, at bimonthly intervals (February, April, June, August, October, and December) across a twelve-month period. The cognitive tasks included two memory tasks: Digit Span and Visual Memory Span from the Wechsler Memory Scale. Additional tasks included a mental rotation task using

the Shephard and Metzler (1971) three-dimensional figures, as well as spatial and verbal versions of a Posner-type hemispheric asymmetry task similar to that used by Corbera (1995).

Additional measures taken at each testing session to confirm the presence of autumn/ winter depressive episodes and associated atypical vegetative symptoms were the Beck Depression Inventory with addendum, Weekly Mood Inventory, and Sleep Quality and Food Preference Questionnaire. The trait version of the Tension and Effort Stress Inventory (Apter, 1988) was also administered at each testing session to determine any differences between the groups, or changes across the twelve-month period, to emotions.

Trend analyses were used specifically to estimate any seasonal variation across the twelve month. A significant quadratic trend, with a winter trough indicates seasonality or cognitive impairment for winter; and a winter peak for depression indicates seasonality or increased depression during winter. For the control group, a significant quadratic trend with either a peak or a trough during the winter months indicates seasonality in the general population.

The SAD groups were expected to show autumn/ winter depressive episodes, and associated atypical behavioural symptoms, with the clinical group showing more extreme ratings than the subsyndromal group. The SAD groups were also expected to show a decrease in hypomania symptoms during winter, as compared to spring/ summer. Because more extreme changes to the sleep

symptom were shown for the SAD groups as compared to controls (chapter 4), similar group differences were predicted for the sleep quality scales. For food preference, SADs were expected to prefer carbohydrates during winter, reflecting the carbohydrate-craving symptom. Little change was expected for protein intake for any group across the twelve-month period.

Deficits to cognitive efficiency for the SAD groups during winter may be expected due to lack of motivation, inability to concentrate, fatigue and/ or energy difficulties. Alternatively, any impairment to cognitive processing may result from deficits in the visual pathways and/ or circadian rhythm disruption. Any deficits to both verbal and spatial conditions of the Hemispheric Asymmetry task may assist in understanding hemispheric specialisation in SAD. Deficits may be evident in spatial tasks for SADs in winter due to the competition between spatial processing and negative emotion in the right hemisphere.

The SAD groups may be expected to show greater levels of tension stress, but not effort stress in winter, resulting from their lack of motivation and inability to concentrate. The SAD groups may also be expected to show greater negative emotions during the autumn and winter months than controls.

Method

Participants

The Seasonal Pattern Assessment Questionnaire (SPAQ) (Rosenthal et al., 1984) (Appendix A) was used to screen seasonal change in mood and behaviour. The sum of six (sleep length, social activity, mood, weight, appetite, and energy level) ranging from 0 (no change) to 4 (extremely marked change) gave a Global Seasonal Score (GSS). C-SAD, S-SAD, and control participants were selected according to the criteria outlined in Longitudinal Study 1. Additional C-SAD and S-SAD participants satisfying the selection criteria were obtained by advertising within the University of Tasmania, the local Mercury newspaper, and local media.

Fifty-eight participants completed all six bimonthly testing sessions. There were 21 control participants (GSS $M = 2.52$, $SD = 2.18$, range of 0 - 6), 19 S-SAD participants (GSS $M = 11.68$, $SD = 1.97$, range of 8 - 15), and 18 C-SAD participants (GSS $M = 16.44$, $SD = 2.73$, range of 13 - 22). Mean ages were, for controls, $M = 22.05$, $SD = 8.28$, age range of 18-43; for S-SADs, $M = 27.26$, $SD = 10.47$, age range of 18-48; and for C-SADs, $M = 29.28$, $SD = 9.28$, age range of 18-47. The female to male ratio was 4:1. Many participants completed both Longitudinal Study 1 and Longitudinal Study 2, though there were some exceptions. All participants for Longitudinal Study 2 were right-handed, hence any left-handed participants completed Longitudinal Study 1 only. In addition, some individuals participated in Longitudinal Study 1 only after requesting a willingness to volunteer for six 1-hour sessions. Further, one female participant became pregnant early during the testing period, and completed Longitudinal Study 2 only.

All participants were Tasmanian residents for a minimum of three years prior to completion of the screening questionnaire to control for climatic adjustment at latitude 43° South. Ethical approval for the research was obtained from the University Ethics Committee (Human Experimentation) prior to laboratory testing. Written informed consent (Appendix B) was obtained from participants.

Apparatus

Psychological measurement. All questionnaires used to determine psychological measurement for Longitudinal Study 1 were also used for the current study. The Beck Depression Inventory with Addendum (BDIadd) and the Weekly Mood Inventory (WMI) were administered to enable validation of seasonal depression. A Sleep Quality and Food Preference Questionnaire (SQFP) was administered to determine participants' quality of sleep and any change in food preference. The Tension and Effort Stress Inventory (TESI) was administered to determine experience of stressors, related efforts to cope, the assessment of a range of pleasant as well as unpleasant moods. Chapter 7 outlines a more complete description of each questionnaire.

Cognitive Tasks. Two memory tasks were used; the Digit Span and Visual Memory Span, from the Wechsler Memory Scale-Revised (Wechsler, 1987) which included an attention/concentration component. The Digit Span and Visual Memory Span memory tasks used standard instructions from the Wechsler Memory Scale-Revised Handbook. Both Digit Span and Visual Memory Span consisted of two parts: Digits Forward and Digits Backward for the Digit Span task, and Tapping Forward and Tapping Backward for the Visual Memory Span.

Verbal and spatial versions of a cognitive processing hemispheric asymmetry task were used as a simple Posner-type RT task as well as to determine any differential hemispheric involvement. The hemispheric asymmetry tasks have previously been shown seasonal differences for control participants (Corbera, 1995). Mental rotation was used as a more complex RT task, requiring spatial involvement (Shepard & Metzlar, 1971), with reaction time consisting of the time it took mentally to rotate the objects to match, and then making a same/different decision. Computer versions of both the hemispheric asymmetry and mental rotation tasks were developed in the School of Psychology, University of Tasmania and installed on an PC computer.

The Hemispheric Asymmetry task, consisted of comparing two stimuli presented consecutively and then making a "same" or "different" response. There were two conditions, verbal and spatial, which were administered separately. The following standard instructions were given for the first task, using either "verbal" or "spatial" as appropriate.

"This task consists of comparing two consecutive verbal/spatial stimuli, and then pressing a "same" or "different" response. The task will commence with a dot in the centre of the screen. You are to fixate on this central point. A centred stimuli (word/symbol) will then appear, followed by the central dot, and then a lateralised stimuli (either to the left OR the right). Your task is to compare the two stimuli and respond "same" or "different". The up arrow (↑) button is the "same", and the down arrow (↓) is "different". Any questions? Remember to maintain visual fixation on the central point."

For the second task the standard instructions were:

"This task is the same, except the stimuli are spatial/verbal. Again there will be a practice first. Remember to maintain visual fixation on the central point."

Verbal stimuli were five letter neutral words selected for their moderate frequency and familiarity, along with a low concreteness and imagery. No winter related words were used. The words were presented on a PC computer in 36-font size. The first word was presented in uppercase, and lateralised words in lower-case to prevent comparison by configuration strategies (see Figure 27).

Spatial stimuli were V-shape symbols which were presented in four directions (\wedge , \vee , $>$, and $<$) as well as three orientations (36° , 90° , and 154°) giving a range of 12 possible figures. Figure 1 shows an example of a centralised symbol, followed by left and right lateralised “same”, then “different” stimuli.

Each condition consisted of 12 practice trials, and 100 test trials presented in 4 blocks, with 10 sec breaks between the blocks. Of these, 40 trial pairs were the same, and 60 trial pairs different. The lateralised stimuli were half to the left of the central point, and half to the right. Stimuli were presented in random order, with no more than 3 consecutive “same” or “different” pairs. The stimulus presentation time for both the central stimuli and the lateralised stimuli was 140 ms. The first central point was presented for 2000 ms as a warning signal prior to presentation of the central stimuli in each pair, and the second central point presentation time was 100 ms.

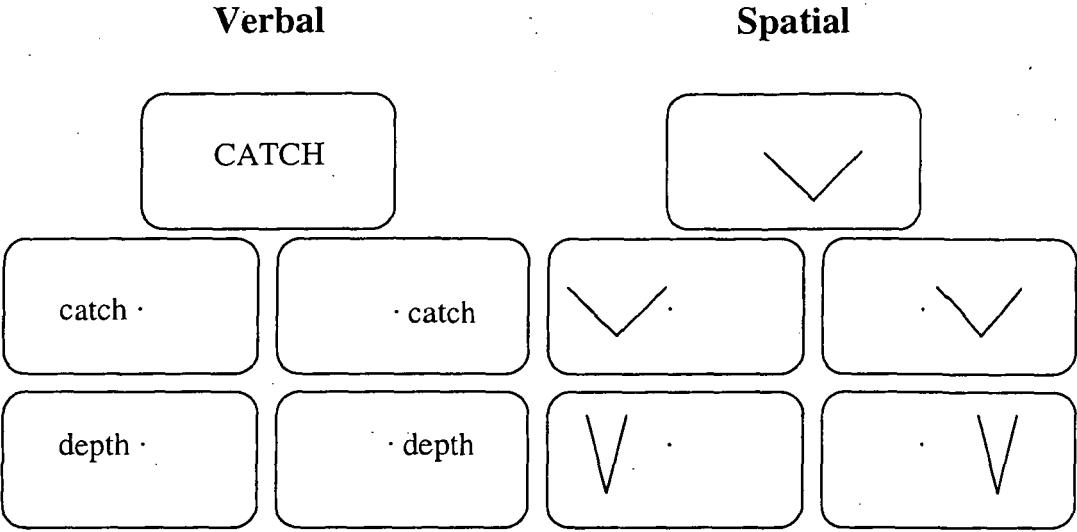


Figure 27. Experimental stimuli for verbal and spatial conditions of the hemispheric asymmetry tasks showing centralised, then left and right lateralised alternatives of “same” and “different” stimuli.

The mental rotation task (Shepard & Metzlar, 1970) consisted of pairs of three-dimensional objects presented on the computer screen with participants required to press a “same” or “different” response. Standard instructions for the mental rotation task were:

“You will be shown pairs of three dimensional objects. They’ll come on the screen together. Your task is to determine whether the two objects have the same three-dimensional shape (even though they may be portrayed in very different orientations), or whether they have inherently different three-dimensional shapes (and so could not be brought into mutual congruence by any rigid motion). The up arrow (↑) button is the “same”, and the down arrow (↓) is “different”. Any questions?”

Participants were also informed that to be the same, the arms of the two objects had to face the same direction after mentally rotating the pairs to match, whereas a mirror image requiring a complete reversal of one figure was “different”.

Participants were also informed that all stimuli consisted of the same number or blocks and it was not helpful to count the blocks.

The stimuli comprised pairs of three-dimensional objects, with each stimulus made up from ten solid cubes (Figure 2). One of each of the three dimensional pairs was rotated either 0°, or in 20° increments to 180°, requiring participants to mentally rotate the object prior to making a response.

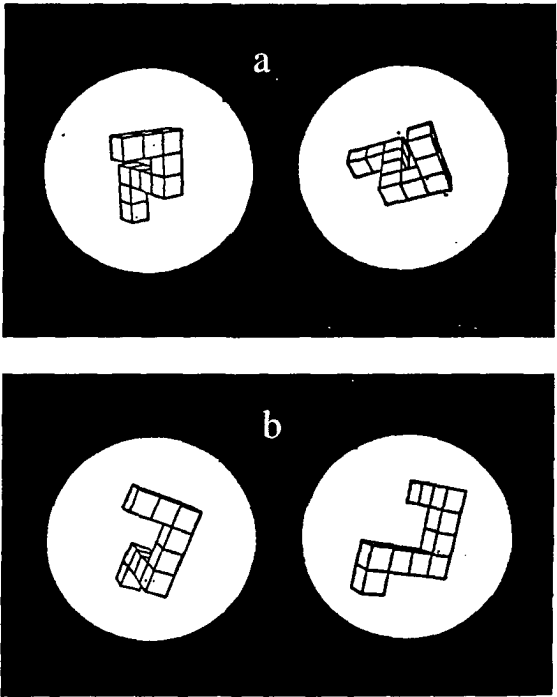


Figure 28. Mental Rotation stimuli comprising pairs of three-dimensional objects showing (a) a *same* pair differing by a 80° rotation, and (b) a *different* pair which cannot be brought into congruence by any rotation

There were 8 practice trials and 54 test trials presented in 2 blocks, with a 10s break between the blocks. Half of the test trial pairs were the same, and half were different. Each trial commenced with a central point as a warning, followed by

presentation of a stimulus pair. The central point presentation time was 10 s and the stimulus presentation time was 100s.

Emotional measurement. The trait version of the TESI (Svebak, 1987) was used to rate the experience of stressors and emotions, and is more fully described in Chapter 7. An additional 7-point scale from the state version of the TESI was included to determine current felt stress. Participants rated the degree of stress (1 = no stress, 7 = high stress) that they were currently feeling.

Procedure

Participants were tested individually. The laboratory was sound attenuated and temperature controlled (24°C). Half of the participants were tested in the morning (8.00 a.m. to 12 noon) and half in the afternoon (2.00 p.m. to 6.00 p.m.) to control for any circadian differences in performance measures. Time of testing was counterbalanced across group and gender. Testing sessions were in February, April, June, August, October, and December. Each participant completed six consecutive sessions. Testing of all participants was completed within a three-year period. Participants were not tested in the first two-week period following the commencement, and the end of the summer daylight savings period.

Each laboratory session commenced with administration of the four questionnaires (BDIadd, WMI, SQPF, & TESI). Participants then completed the cognitive tasks (memory tasks, mental rotation, and hemispheric asymmetry) in counterbalanced order, with each participant completing the tasks in their same given order for all six sessions. The two memory tasks, Digit Span and Visual

Memory Span, were counterbalanced within memory task, and the verbal and spatial conditions of hemispheric asymmetry were also counterbalanced. Standard instructions were given for each task and practice allowed. The experimenter ensured the participant understood the task prior to commencement.

For the Hemispheric Asymmetry and Mental Rotation tasks, half of the participants in each group used their right forefinger for the up arrow (↑) button or “same” response, and their left forefinger for the down arrow (↓) button or “different” response. The other half used the reverse forefingers.

Design

Laboratory testing commenced with twelve participants (four control, four S-SAD, and four C-SAD). Four additional participants were added to each group at each consecutive bimonthly laboratory testing period, with each participant completing six testing sessions. Due to the longitudinal nature of the experiment, many participants were unable to complete their six bimonthly sessions.

Replacement participants were added at the next opportunity. The difficulty of retaining SAD participants, particularly through their ‘at risk’ period was greater than that of control participants.

All dependent variables used a 6 x 3 (Month x Group) mixed between and within subjects design. The between groups factor was Group (Control, S-SAD, and C-SAD). The within groups factor for all dependent variables was Month (February, April, June, August, October, and December).

The dependent measures for depression and behavioural symptoms were BDIadd, four scales for the WMI (DMI, TVS, AVS, and HMI), four scales for the SQFP (good nights/ bad nights, restful sleep/ disturbed sleep, slept lightly/ slept deeply, and felt refreshed/ felt fatigued). The BDIadd and WMI scales score was the sum of the item ratings for each subscale. Food preference was the percentage of each group experiencing a change in food preference. All other questionnaire scales used mean item ratings.

For Digit Span and VMS, the dependent measure was the number of correct items in each forward and backward condition. For Mental Rotation and Hemispheric Asymmetry the dependent measure was RT. Each of the two Hemispheric Asymmetry versions (verbal and spatial) had two conditions (LVF and RVF).

Emotional analyses used trait TESI scales which were four sources of Tension Stress (work, family, finance, own body), four sources of Effort Stress (work, family, finance, own body), and four groupings of the sixteen emotions, positive somatic (relaxation, excitement, placidity, and provocativeness), negative somatic (anxiety, boredom, anger, and sullenness), positive transactional (pride, modesty, gratitude, and virtue), and negative transactional (humiliation, shame, resentment, and guilt). The state TESI current felt stress scale was also used. Each of the TEST groupings used averaged rating. All TESI items used rating.

Data Scoring and Analyses

Trend analyses using planned polynomial contrasts were conducted for Month, and Month x Group, on each dependent variable specifically to estimate any

quadratic and/ or cubic seasonality trend across the twelve-month period (Maxwell & Delaney, 1989). Where polynomial contrasts revealed a significant quadratic or cubic interaction for Month x Group, polynomial contrasts were conducted for each group individually to locate the significant seasonality effect.

Repeated measures MANOVAs using Pillai's criterion were also performed on all dependent variables, except for the TESI analyses. Where multivariate analyses revealed a significant main effect or interaction, follow up with appropriate univariate analyses conducted as described in Chapter 7. The Type 1 error rate was $\alpha = .05$, except where otherwise indicated.

Because of the large number of repeated measures in the TESI relative to numbers of participants in groups, ANOVAs were used for all TESI analyses and epsilon adjustment using the Greenhouse Geiser correction procedure for repeated measures (Vasey & Thayer, 1987).

All analyses for the screening questionnaire, depression and behavioural symptoms, and emotion analyses were conducted as described for Longitudinal Study 1 (Chapter 7). For the TESI, main effects for emotions and stressors are meaningless and will not be described: however, interactions involving emotions and stressors are important and will be interpreted.

Cognitive tasks. Digit Span, and Visual Memory Span both used a 6 x 3 (Month x Group) repeated measures MANOVA for each condition: forward and

backward. Hemispheric Asymmetry used separate 6 x 2 x 3 (Month x visual field [left/ right] x Group) repeated measures MANOVA for each condition: verbal and spatial. Mental Rotation was analysed using a 6 x 3 (Month x Group) repeated measures MANOVA.

Results

Screening questionnaire

Means and standard deviations for self-rated scores from the Seasonal Pattern Assessment Questionnaire (SPAQ) are presented in Table 26.

Univariate analyses. One way between groups ANOVAs were performed for Global Seasonality Score (GSS), Global Rating (GR), Weight Fluctuation (WtFluct), and Age. Main effects were revealed for GSS, GR, and WtFluct.

The main effect for GSS, $F(2, 55) = 186.03, p < .001$, indicated highly significant differences between the groups. LSD post hoc analyses showed highly significant differences between all pair-wise group comparisons at the $\alpha = .001$ level of significance. Table 26 shows C-SADs to have a higher GSS than S-SADs and controls. S-SADs also have a higher GSS than controls.

The main effect for GR, $F(2, 55) = 27.54, p < .001$, indicated highly significant differences between the groups. LSD post hoc analyses showed significant differences between all pair-wise group comparisons. Table 26 also shows C-SADs to have a higher GR than S-SADs and controls at the $p = .001$ level of significance. S-SADs are also higher than controls for GR.

Table 26

Means and Standard Deviations for Scores on the Screening Questionnaire (Seasonal Pattern Assessment Questionnaire) (SPAQ) for Control, Subsyndromal SAD, and Clinical SAD Groups in Longitudinal Study 2 of Cognitive Performance

	Source	Control		Subsyndromal SAD		Clinical SAD	
		Mean	SD	Mean	SD	Mean	SD
Degree of seasonal change to:	Appetite	0.24	0.44	1.58	0.77	2.78	0.81
	Energy	0.48	0.60	2.37	0.76	3.22	0.55
	Mood	0.52	0.68	2.42	0.69	3.06	0.73
	Sleep	0.62	0.59	2.16	0.60	2.39	0.78
	Social	0.62	0.59	2.16	0.60	2.39	0.78
	Weight	0.24	0.44	1.21	0.63	2.33	0.84
Global Seasonality Score		2.52	2.18	11.68	1.97	16.44	2.73
Global Rating		0.00	0.22	0.74	1.28	2.39	1.29
Weight Fluctuation		1.48	0.87	2.05	1.03	2.72	1.56
Sleep Length	Autumn	8.21	1.17	8.16	1.50	8.94	1.51
	Winter	8.33	1.20	9.00	1.45	10.11	1.67
	Spring	8.14	1.20	7.79	0.98	8.22	1.35
	Summer	7.83	1.49	6.63	1.26	7.50	1.44
Age		22.05	8.28	27.26	10.47	29.28	9.89

Note. The degree of change in a twelve month period irrespective of season for each symptom ranged from 0 (no change) to 4 (extremely marked change). Global Seasonality Score was the sum of the six symptoms. Global Rating was from 0 (not a problem) to 5 (disabling). Weight Fluctuation in a twelve month period was recorded on a scale from 1 to 6 where 1 = 0-2 kg, 2 = 2-3 kg, 3 = 4-5 kg, 5 = 8-10 kg, 6 = over 10 kg. Sleep length was the approximate number of hours of sleep per 24 hour period during each season.
n = 21 Control, 19 Subsyndromal SAD, and 18 Clinical SAD participants. All participants completed the SPAQ during the early autumn.

The main effect for WtFluct, $F(2, 55) = 5.46, p = .007$, indicated significant differences between the groups. LSD post hoc analyses showed the weight of C-SADs to fluctuate significantly more than controls with no other significant differences. This difference in WtFluct between C-SADs and controls can be seen from Table 26. The one-way ANOVA for age was non-significant.

Degree of change across seasons. A one-way between groups MANOVA was performed on six dependent variables measuring the degree of change across the seasons to the six symptoms: appetite, energy, mood, sleep, socialisation, and weight. The MANOVA was highly significant, Pillai's Trace = 1.08, $F(12, 102) = 10.07, p < .001$. Subsequent univariate F tests on each dependent variable showed all symptoms to contribute to significant differences between the groups. Univariate F values were: appetite, $F(2, 55) = 67.67, p < .001$; energy, $F(2, 55) = 94.29, p < .001$; mood, $F(2, 55) = 70.67, p < .001$; sleep, $F(2, 55) = 42.85, p < .001$; socialisation, $F(2, 55) = 41.60, p < .001$; and weight, $F(2, 55) = 50.95, p < .001$. Subsequent LSD post hoc analyses showed significant differences between all pairwise group comparisons for all symptoms, except sleep, at the $\alpha = .01$ level of significance. Figure 29 shows these differences between the groups for each symptom. From Figure 29, C-SADs rate the symptoms higher than S-SADs and controls. S-SADs symptom ratings are also higher than controls. For sleep, C-SAD and S-SAD groups rated sleep change significantly higher than controls at the $\alpha = .001$ level of significance, but did not differ significantly from each other.

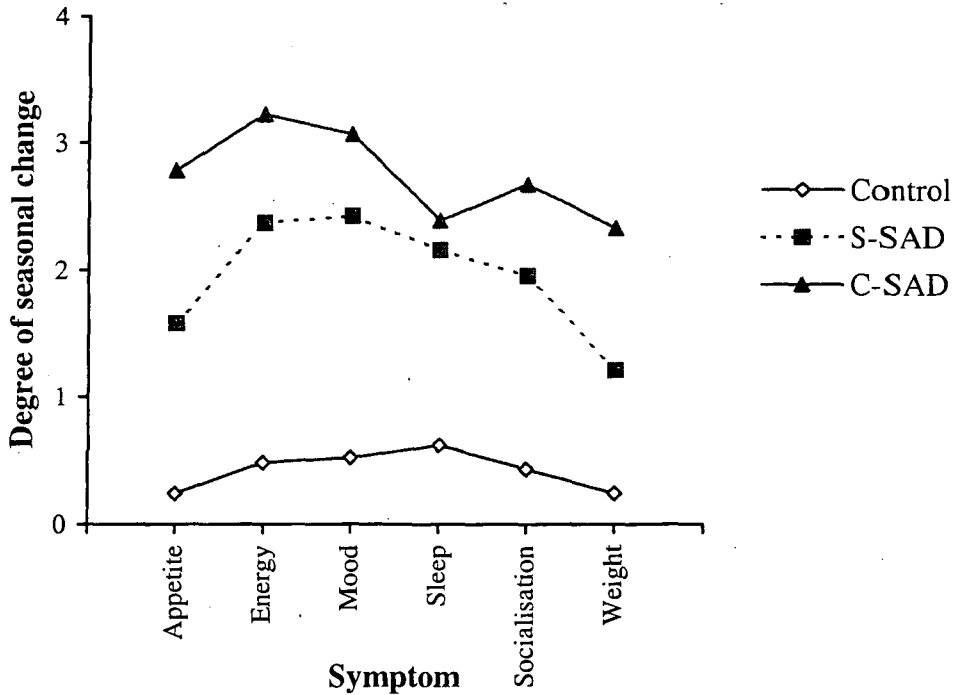


Figure 29. Degree of seasonal change for control, subsyndromal SAD (S-SAD), and clinical SAD (C-SAD) groups for six symptoms on the Seasonal Pattern Assessment Questionnaire

Hours of sleep per twenty four hour period. A 3 x 4 (Group x Season) repeated measures MANOVA revealed a main effect for Season, and a significant Group x Season interaction. The significant main effect for Season, Pillai's Trace = 0.55, $F(3, 53) = 21.45$, $p < .001$, indicated significant differences between the seasons. The significant Group x Season interaction, Pillai's Trace = 0.38, $F(6, 108) = 4.22$, $p = .001$ indicated the groups differed in their hours of sleep across the seasons. Figure 30 shows these differences. Subsequent univariate between groups analyses for each season separately showed significant differences for winter, $F(2, 55) = 6.62$, $p = .003$, and for summer, $F(2, 55) = 7.55$, $p = .024$. For winter, C-SADs slept significantly more hours than S-SADs and controls, while for summer, S-SADs slept significantly less hours than controls (Figure 30).

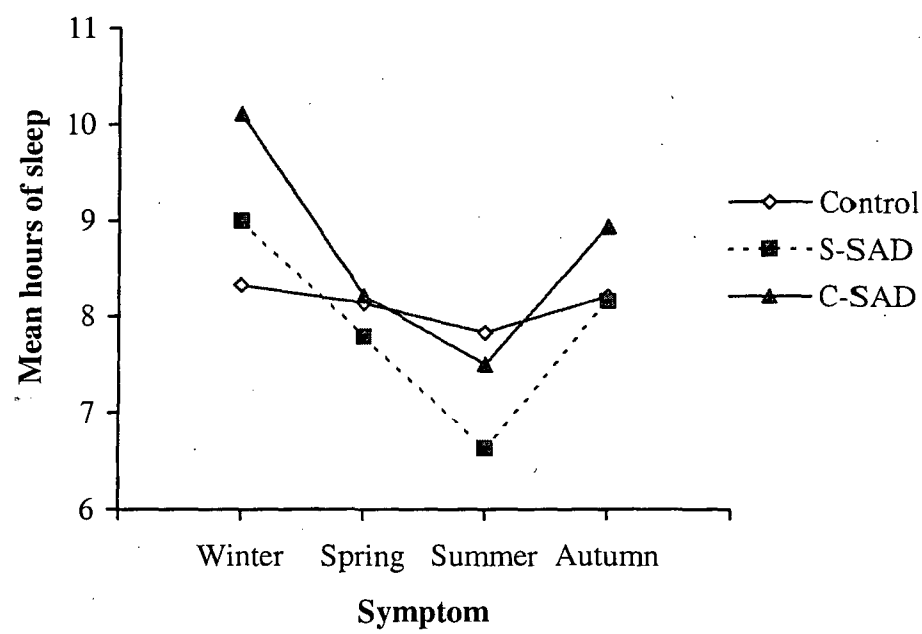


Figure 30 . Mean hours of sleep for each season as retrospectively self-rated by Control, Subsyndromal SAD (S- SAD), and Clinical SAD (C-SAD) groups on the Seasonal Pattern Assessment Questionnaire

Univariate repeated measures ANOVAs were also conducted across the seasons for each group separately. Significant differences across the seasons were revealed for C-SADs, $F(3, 15) = 9.01$, $p = .001$, and for S-SADs, $F(3, 16) = 13.62$, $p < .001$. Subsequent post hoc analyses between pair-wise comparisons show both C-SADs and S-SADs reported greater hours of sleep for winter, than for spring, summer and autumn.

Summary of SPAQ results. Overall, the SPAQ differentiates between the groups for GSS, GR, WtFluct, degree of change to each symptom: appetite, energy, mood, sleep, socialisation, and weight, and hours of sleep in winter only, with the

SAD groups rating higher than controls for all variables. C-SADs also rated higher than S-SADs for all variables except WtFluct and degree of change to the sleep symptom. The SAD groups also slept more in winter as compared to spring, summer, and autumn.

Depression and behavioural symptoms

Mean self-rated depression and behavioural symptom scores and standard deviations for BDIadd and WMI are presented in Table 27.

Beck Depression Inventory with Addendum. For BDIadd, a trend analysis was conducted using planned polynomial contrasts revealing significant quadratic and cubic effects for month, and a significant cubic interaction between month and group.

The trend analysis showed significant quadratic, $F(1, 54) = 9.33, p = .004$, and cubic, $F(1, 54) = 19.19, p < .001$, polynomial effects for month showing that overall, participants experience seasonality for the BDIadd with a peak in depression in the autumn/ winter months. Trend analysis also showed a significant cubic interaction between month and group, $F(2, 54) = 5.18, p = .009$. Figure 31 shows this cubic interaction. Subsequent polynomial contrasts conducted on each group separately revealed a significant cubic effect for C-SADs, $F(1, 16) = 12.62, p = .003$, while S-SADs showed both a significant quadratic, $F(1, 18) = 5.21, p = .035$, and a cubic effect, $F(1, 18) = 6.19, p = .023$. Controls did not show any significant effects. Figure 31 reflects these significant effects for the SAD groups showing seasonal peaks for depression in April and June.

Table 27

Means and Standard Deviations for Depression and Behavioural Symptoms (Beck Depression Inventory with Addendum and Weekly Mood Inventory) for Control, Subsyndromal SAD, and Clinical SAD Groups at Bimonthly Testing Sessions

	Group	February		April		June		August		October		December	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Beck Depression Inventory with Addendum	Control	2.57	5.12	3.43	6.98	3.19	6.45	2.43	4.95	2.90	4.97	2.12	4.69
	S-SAD	3.74	4.81	6.84	7.07	7.79	7.08	5.26	4.74	5.11	6.77	4.37	6.28
	C-SAD	7.76	9.90	13.88	10.29	12.06	7.53	9.00	5.98	7.35	6.21	7.47	6.87
Weekly Mood Inventory													
Depressed Mood and Ideation (6 items)	Control	7.38	7.31	6.95	8.35	9.24	7.56	5.05	5.94	7.86	6.46	6.38	6.28
	S-SAD	8.89	6.41	12.05	7.43	11.05	6.30	8.89	5.57	8.42	6.22	8.95	7.29
	C-SAD	11.53	9.24	16.71	9.04	15.82	6.94	11.65	6.62	10.71	7.10	10.00	7.42
Typical Vegetative Symptoms (5 items)	Control	7.00	6.12	5.52	5.94	8.62	6.42	5.57	6.01	7.95	5.51	6.52	5.10
	S-SAD	9.32	5.09	10.84	6.76	9.21	7.20	6.37	5.42	7.47	6.76	9.00	5.06
	C-SAD	12.65	7.60	13.35	8.12	10.29	7.11	10.53	4.99	12.53	6.39	14.06	5.58
Atypical Vegetative Symptoms (5 items)	Control	8.76	3.32	9.00	3.91	9.52	4.52	8.81	4.76	9.14	5.02	8.86	3.34
	S-SAD	10.21	4.91	12.58	5.81	13.00	6.20	12.95	6.22	10.26	5.34	10.26	4.76
	C-SAD	10.06	6.55	14.06	5.62	15.94	7.80	15.41	6.08	12.00	3.69	9.18	4.54
Hypomania Mood and Ideation (10 items)	Control	35.76	11.70	34.95	11.24	35.29	9.91	36.67	9.50	35.14	8.45	35.71	10.07
	S-SAD	35.00	8.45	30.00	6.20	30.26	9.43	31.16	8.83	33.47	8.85	36.11	8.14
	C-SAD	35.65	10.35	24.71	9.59	26.18	10.48	30.82	10.28	30.94	11.04	34.12	11.30

Note. Weekly Mood Inventory ratings were made on a 7-point scale (0 = not at all, 6 = very much) and totalled for each factor.

n = 21 Control, 19 Subsyndromal SAD (S-SAD), and 18 Clinical SAD (C-SAD) participants

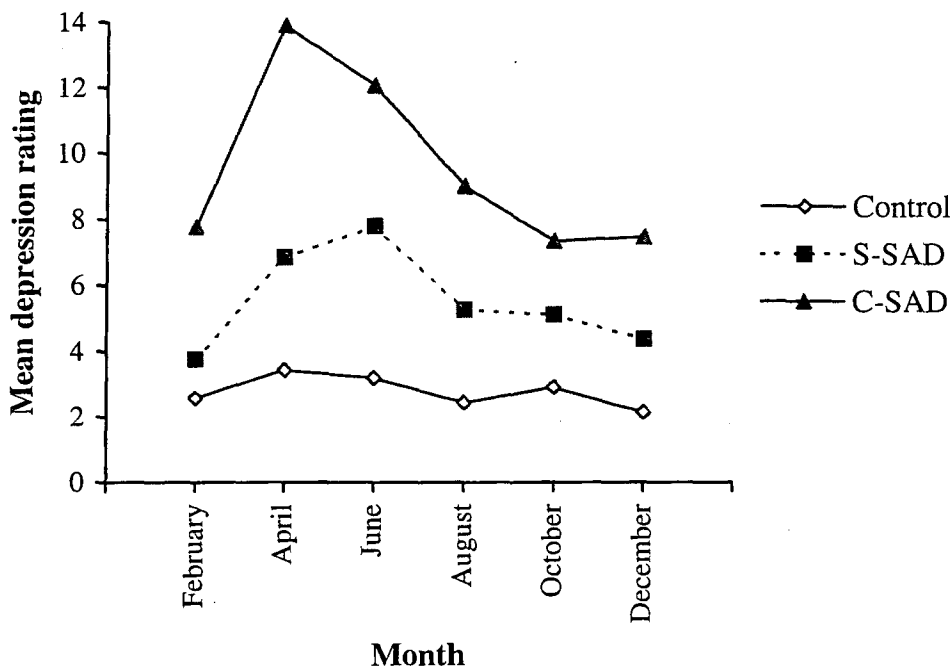


Figure 31. Depression ratings from the Beck Depression Inventory with Addendum for Control, Subsyndromal SAD (S-SAD), and Clinical SAD (C-SAD) groups for depression at bimonthly intervals for a twelve-month period

A 6 x 3 (Month x Group) repeated measured MANOVA was also performed revealing significant main effects for month, and for group. The main effect for Month, Pillai’s Trace = .30, $F(5, 50) = 4.30$, $p = .003$, indicated a significant difference across the months. Post hoc pair-wise comparisons showed significantly higher depression in April and June than in any other month at the $p = .01$ significance level (see Table 27). The main effect for group, $F(2, 54) = 8.70$, $p = .001$ indicated the groups differed in depression rating, regardless of month. LSD post hoc pair-wise comparisons showed C-SADs to rate depression significantly higher than S-SADs and controls (see Table 27).

Weekly Mood Inventory. For WMI, trend analyses using planned polynomial contrasts were performed for each subscale: depressed mood and ideation (DMI), typical vegetative symptoms (TVS), atypical vegetative symptoms (AVS), and hypomania mood and ideation (HMI). Overall, for DMI, trend analyses using polynomial contrasts revealed significant quadratic and cubic main effects for month, as well as a significant cubic interaction between month and group. For AVS, and for HMI, trend analyses revealed significant quadratic main effects for month, as well as interactions between month and group.

Following the trend analyses, 6 x 3 (Month x Group) repeated measures MANOVAs using Pillai's Trace criterion were also conducted for each subscale of the WMI. Overall, the MANOVAs revealed significant main effects for Month for all four subscales, and for Group, for DMI, TVS, and AVS only.

For DMI, there were significant quadratic, $F(1, 54) = 4.02, p = .05$, and cubic, $F(1, 54) = 16.04, p < .001$ main effects for month indicating an overall seasonal pattern across the twelve month period. A significant cubic interaction between month and group for DMI, $F(2, 54) = 3.17, p = .05$, indicated the groups varied in their seasonal pattern across the twelve month period. Subsequent polynomial contrasts conducted for each group separately revealed significant seasonal effects for both SAD groups, though not for controls. C-SADs and S-SADs showed significant cubic effects, $F(1, 16) = 9.14, p = .008$, and $F(1, 18) = 9.31, p = .007$, respectively.

From the 6 x 3 (Month x Group) MANOVA, DMI revealed a main effect for month, Pillai's Trace = .30, $F(5, 50) = 4.35, p = .002$. Subsequent LSD post hoc

pair-wise comparisons showed DMI to be rated significantly higher in April and June than all other months. MANOVA also revealed a main effect for group, $F(2, 54) = 5.31, p = .008$. C-SADs rated DMI significantly higher than controls.

For TVS, trend analyses using polynomial contrasts did not reveal any quadratic or cubic main effects or interactions. From the 6×3 (Month \times Group) MANOVA, TVS revealed a main effect for month, Pillai's Trace = .22, $F(5, 50) = 2.78, p = .027$. Post hoc pair-wise comparisons revealed non-seasonal typical depression symptoms to be significantly *lower* in August than in all other months except June. MANOVA also revealed a main effect for group, $F(2, 54) = 7.26, p = .002$. C-SADs rated TVS higher than S-SADs and controls.

For AVS, the significant quadratic main effect for month, $F(1, 54) = 21.58, p < .001$, indicated an overall seasonal pattern across the twelve month period. A significant cubic interaction between month and group for AVS, $F(2, 54) = 6.33, p = .003$, indicated the groups varied in their seasonal pattern across the twelve month period. Subsequent polynomial contrasts conducted for each group separately revealed significant quadratic effects for C-SADs, $F(1, 16) = 15.68, p = .001$, and S-SADs, $F(1, 18) = 8.28, p = .01$.

From the 6×3 (Month \times Group) MANOVA, AVS revealed a main effect for month, Pillai's Trace = .33, $F(5, 50) = 4.88, p = .001$. AVS is rated significantly higher in April, June, and August, than in February and December ($p < .01$). MANOVA also revealed a main effect for group, $F(2, 54) = 6.14, p = .004$. C-SADs and S-SADs rated AVS significantly higher than controls.

For HMI, the significant quadratic, $F(1, 54) = 11.13, p = .002$, and cubic, $F(1, 54) = 9.04, p = .004$, main effect for month indicated an overall seasonal pattern across the twelve month period. A significant quadratic interaction between month and group for HMI, $F(2, 54) = 3.19, p = .049$, indicated the groups varied in their seasonal pattern across the twelve month period. Subsequent polynomial contrasts conducted for each group separately revealed significant quadratic $F(1, 16) = 6.18, p = .024$, and cubic, $F(1, 18) = 7.18, p = .016$, effects for C-SADs, and a quadratic effect for S-SADs, $F(1, 18) = 7.24, p = .015$.

From the 6 x 3 (Month x Group) MANOVA, HMI revealed a main effect for month, Pillai's Trace = .31, $F(5, 50) = 4.46, p = .002$. HMI is rated significantly lower in April than in all other months except June. HMI is also lower in June than in February and December at the $\alpha = .01$ significance level. These differences can be seen in Table 27.

Figure 32 reflects the seasonality revealed from trend analyses conducted on each of the WMI subscales. DMI and AVS show a peak, and HMI shows a trough for the autumn/ winter months. No significant effects were found for the TVS subscale, or for the control group in any subscale.

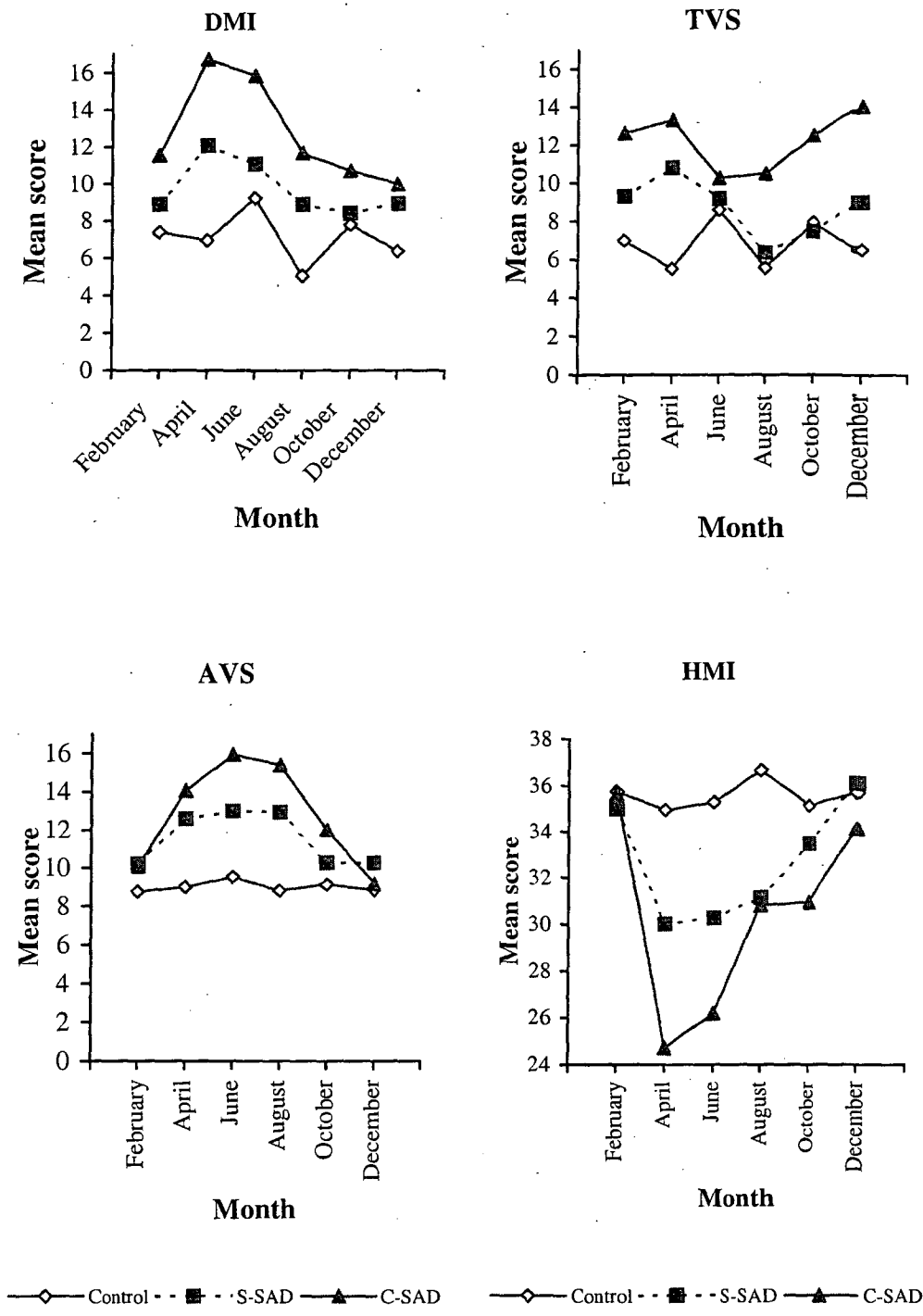


Figure 32. Weekly Mood Inventory Subscales: Depressed Mood and Ideation (DMI) (6 items), Typical Vegetative Symptoms (TVS) (5 items), Atypical Vegetative Symptoms (AVS) (5 items), and Hypomania Mood and Ideation (HMI) (10 items) for control, subsyndromal SAD (S-SAD), and clinical SAD (C-SAD) groups at bimonthly sessions over a twelve-month period.

Summary of results for depression and behavioural symptoms. Significant quadratic and cubic curves representing seasonality were shown for the SAD groups reflecting increases in BDIadd, and WMI subscales for depressed mood and ideation (DMI), and atypical vegetative symptoms (AVS), as well as a decrease in hypomania mood and ideation (HMI), for the SAD groups during the autumn/ winter months. C-SADs rated BDIadd and the WMI subscale for typical vegetative symptoms (TVS) higher than S-SADs, though did not differ for DMI and AVS.

Sleep Quality and Food Preference Questionnaire. Mean self-rated sleep quality scores and standard deviations from four subscales: good nights/ bad nights, restful nights/ disturbed nights, slept lightly/ slept deeply, and felt refreshed/ fatigued, are shown in Table 28.

For sleep quality, planned polynomial contrasts were performed separately for each of the four sleep quality subscales. For the good/ bad nights scale, trend analysis revealed a significant cubic main effect for month, and a significant cubic interaction between month and group. For the refreshed/ fatigued scale, trend analysis revealed a significant cubic main effect. Trend analyses for the restful/ disturbed nights and slept lightly/ deeply subscales were non-significant.

Table 28

Mean Ratings and Standard Deviations for Four Sleep Quality Scales: Good/ Bad Nights, Restful/ Disturbed Nights, Slept Lightly/ Deeply, and Felt Refreshed/ Fatigued, for Control, Subsyndromal SAD, and Clinical SAD Groups at Bimonthly Testing Sessions

Subscale	Group	February		April		June		August		October		December	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Good/ bad nights	Control	2.62	1.47	2.29	1.42	3.10	1.64	2.67	1.65	2.86	1.49	2.43	1.50
	S-SAD	3.00	1.25	3.95	1.65	3.11	1.52	2.47	1.02	2.53	1.31	3.11	1.33
	C-SAD	3.88	2.50	4.71	1.86	3.53	1.55	3.82	1.63	3.47	2.07	4.18	1.42
Restful/ disturbed nights	Control	2.67	1.46	2.29	1.23	2.57	1.47	2.38	1.69	2.76	1.45	2.48	1.40
	S-SAD	3.42	1.46	3.53	1.90	3.32	1.83	2.79	1.32	2.37	1.50	2.95	1.75
	C-SAD	4.06	2.22	4.59	2.00	3.59	1.62	3.71	1.61	3.71	2.28	4.06	1.64
Slept lightly/ deeply	Control	5.05	1.72	5.19	1.60	4.95	1.49	4.71	1.79	5.33	1.32	5.33	1.46
	S-SAD	4.21	1.69	4.53	1.54	4.63	1.77	5.00	1.49	5.37	1.46	4.32	1.70
	C-SAD	4.65	1.80	3.65	2.03	4.12	1.80	4.65	1.62	3.88	2.03	4.24	1.56
Felt refreshed/ fatigued	Control	3.19	1.60	3.24	1.87	3.57	1.69	3.24	1.81	3.48	1.66	3.43	1.16
	S-SAD	3.74	1.56	4.00	1.63	4.00	1.56	3.74	1.45	3.16	1.71	3.74	1.66
	C-SAD	4.12	2.09	5.24	1.52	4.65	1.58	4.41	1.70	4.29	2.11	3.59	1.80

Note. Ratings were made on four 7-point scales where 1 = good nights, 7 = bad nights; 1 = restful nights, 7 = disturbed nights; 1 = slept lightly, 7 = slept deeply; and 1 = felt refreshed, 7 = felt fatigued.

n = 21 Control, 19 Subsyndromal SAD (S-SAD), and 18 Clinical SAD (C-SAD) participants.

Repeated measures 6 x 3 (Month x Group) MANOVAs were also performed for each sleep quality subscale. Overall, for the good nights/ bad nights scale, main effects were revealed for month, and for group, as well as a significant interaction between month and group. Main effects for group were also revealed for the three remaining scales: restful nights/ disturbed nights, slept lightly/ slept deeply, and felt refreshed/ felt fatigued.

For the good/ bad nights scale, polynomial contrasts revealed a significant cubic main effect for month, $F(1, 54) = 8.99, p = .004$, indicating a seasonal pattern across the months. The good/ bad nights subscale also showed a significant cubic interaction between month and group, $F(2, 54) = 5.87, p = .005$, which indicated the groups varied in their seasonal pattern across the months. Subsequent polynomial contrasts for each group separately revealed a significant cubic effect for S-SADs only, $F(1, 18) = 21.71, p < .001$. Figure 33 shows this seasonal trend for S-SADs to experience worse nights for April, with a secondary peak for December. Figure 33 also shows SADs to experience better nights for August and October.

For the good/ bad nights scale, MANOVA revealed main effects for month, Pillai's Trace = 0.23, $F(5, 50) = 2.87, p = .024$, and for group, $F(2, 54) = 7.75, p = .001$. A significant interaction between month and group, Pillai's Trace = 0.38, $F(10, 102) = 2.41, p = .013$, indicated the groups varied in their ratings across the months. Subsequent univariate analyses between the groups for each month separately revealed significant differences for all months except June and October. C-SADs rated their sleep quality significantly worse than controls for all months

except June and October. For August and December, C-SADs also rated worse than S-SADs. For April, S-SADs rate worse than controls. Repeated measures ANOVAs were conducted across the months for each group separately revealing significant differences for S-SADs only. S-SADs had significantly worse nights in April than in all other months except June. Figure 33 shows these group differences.

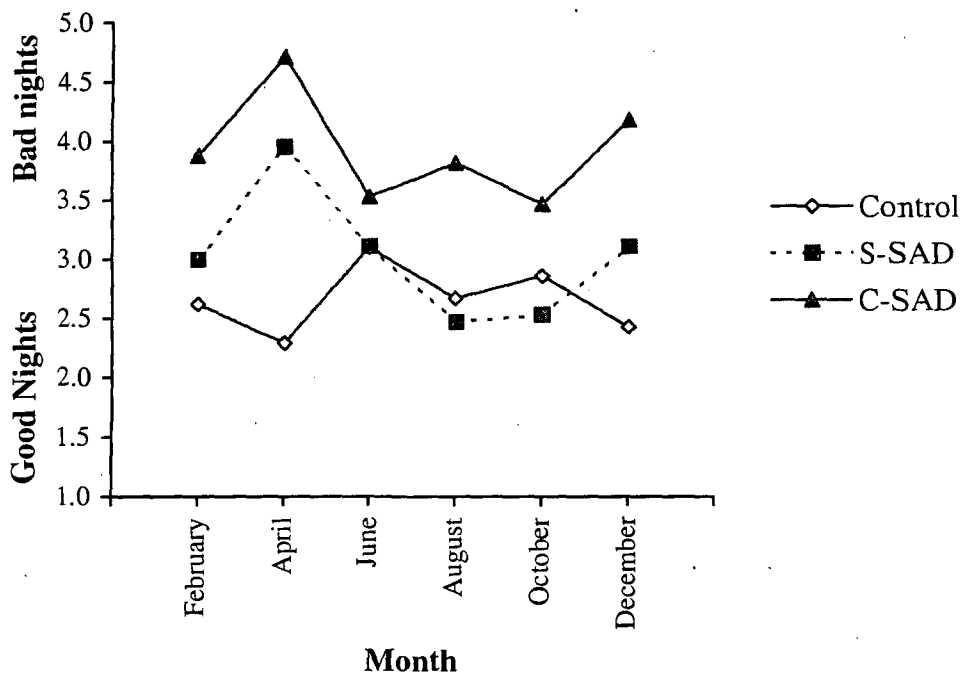


Figure 33. Sleep quality subscale (1 = good nights, 7 = bad nights) ratings for control, subsyndromal SAD (S-SAD) and clinical SAD (C-SAD) groups at bimonthly sessions

For the restful/ disturbed nights scale, MANOVA revealed a main effect for group, $F(2,54) = 11.08, p < .001$, indicating significant differences between the groups. C-SADs rated significantly more disturbed nights than S-SADs and controls at the $p = .01$ significance level.

For the slept lightly/ deeply scale, MANOVA revealed a main effect for group, $F(2, 54) = 2.54, p = .025$, indicating significant differences between the groups. C-SADs slept lighter than controls at the $p = .01$ significance level.

For the refreshed/ fatigued scale, polynomial contrasts revealed a significant cubic main effect for month, $F(1, 54) = 4.68, p = .035$, indicating a seasonal pattern across the months. Figure 34 shows participants to feel more fatigued for April and June. For the refreshed/ fatigued scale, MANOVA revealed a main effect for Group, $F(2, 54) = 3.31, p = .044$, indicating significant differences between the groups (Table 28). C-SADs felt more fatigued than controls.

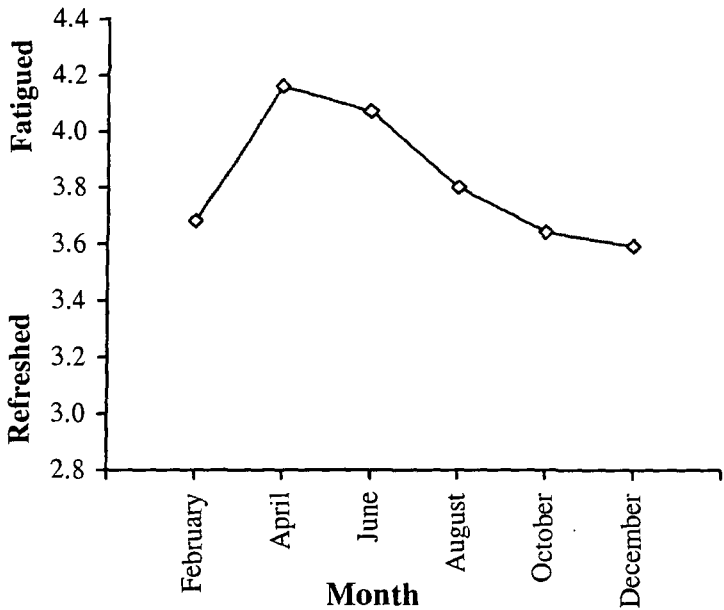


Figure 34. Seasonal pattern for participants overall for the felt refreshed/ fatigued Sleep Quality subscale

For food preference, the percentage of each group who experienced a change for each of the four choices for each month was calculated, and is presented in Table 29. Table 29 shows higher percentages of the SAD groups to record an

Table 29

Percentage of each Group (Control, Subsyndromal SAD, and Clinical SAD) Recording Changes in Food Preference (Fats, Protein, Carbohydrates,) at Bimonthly Intervals

		February	April	June	August	October	December
Preference	Group	Percentage of Group					
Fats	Control		9.52	4.76	9.52		
	S-SAD	5.26	21.05	10.53	21.05	5.26	5.26
	C-SAD	17.65	35.29	47.06	29.41	5.26	11.76
Protein	Control			9.52		4.76	
	S-SAD	5.26	10.53	5.27		5.26	
	C-SAD	5.88	5.88	5.26			11.76
Carbohydrates	Control	14.29	4.76	23.81	14.29		4.76
	S-SAD	10.53	10.53	21.06	42.11	21.05	
	C-SAD	29.41	29.41	47.06	35.29	17.65	11.76

Note. $n = 21$ Control, 19 Subsyndromal SAD (S-SAD), and 17 Clinical SAD (C-SAD) participants. Missing values represent a particular group having no participants recording changes in food preference for the month.

increase in preference for fats and carbohydrates during the autumn/ winter months. This trend is not evident for the spring/ summer months. Table 29 shows controls to record an increase in carbohydrates for June. Little variation is evident for protein or other foods for any month.

Summary of Sleep Quality and Food Preference Results. Significant quadratic curves representing seasonality was evident for S-SADs only for the good/ bad nights scale. Seasonality was also shown for felt refreshed/ fatigued scale reflecting fatigue for April and June. The groups varied in their ratings for the good/ bad nights scale with C-SADs experiencing worse nights of sleep for all months than S-SADs and controls for all months except June and October. Group differences were evident in the three remaining subscales. C-SADs had more disturbed nights than S-SADs and controls, and also slept lighter and felt more fatigued than controls. The SAD groups showed a tendency to prefer fats and carbohydrates during the autumn/ winter months.

Cognitive Tasks

Means and standard deviations for the cognitive tasks are presented in two tables. The memory tasks, Digit Span and Visual Memory Span are presented in Table 30, while Mental Rotation and Hemispheric Asymmetry are presented in Table 31.

Table 30

Means and Standard Deviations for Memory Tasks (No. of Items Correct for Digit Span and Visual Memory Span from the Wechsler Memory Scale) for Control, Subsyndromal SAD, and Clinical SAD Groups at Bimonthly Testing Sessions

			February		April		June		August		October		December	
			Group	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Memory Tasks (No. of items correct)														
Digit Span	Forward	Control	9.38	1.91	9.05	2.13	9.48	1.69	8.86	1.93	8.71	1.65	9.57	2.01
		S-SAD	9.26	2.02	9.32	2.06	9.26	2.08	9.47	2.09	9.53	2.01	9.47	2.29
		C-SAD	9.06	1.48	9.12	1.73	8.71	1.45	8.59	1.62	9.41	1.80	9.00	1.94
	Backward	Control	7.33	2.01	7.10	2.07	6.81	1.91	7.14	2.01	6.90	1.92	7.10	1.70
		S-SAD	6.74	2.38	6.89	2.02	6.74	2.45	7.26	2.26	6.79	1.72	7.58	2.48
		C-SAD	7.53	2.00	7.24	2.11	6.53	1.62	7.12	2.18	7.18	2.13	6.82	2.27
Visual Memory Span	Forward	Control	9.10	2.02	9.52	1.94	9.10	1.45	9.57	1.96	8.62	1.43	9.29	1.85
		S-SAD	8.53	1.93	8.58	1.54	8.79	1.78	8.74	1.73	8.37	2.14	9.37	2.19
		C-SAD	7.41	2.21	7.35	1.32	7.29	1.86	7.76	1.30	7.82	1.38	7.47	1.81
	Backward	Control	9.19	1.40	9.48	1.57	9.10	1.26	8.86	1.71	9.00	1.18	8.81	1.40
		S-SAD	8.58	1.74	8.16	1.34	8.21	1.72	8.11	1.33	8.42	1.54	8.32	1.45
		C-SAD	7.65	1.41	7.65	1.62	7.71	1.36	8.12	1.11	7.53	1.46	7.35	1.41

Note. $n = 21$ Control, 19 Subsyndromal SAD (S-SAD), and 17 Clinical SAD (C-SAD) participants.

Table 31

Means and Standard Deviations for Mental Rotation and Hemispheric Assymetry Tasks (Response Time ms.) for Control, Subsyndromal SAD and Clinical SAD groups at Bimonthly Testing Sessions

		February			April		June		August		October		December	
		Group	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Mental Rotation (RT ms.)														
		Control	2593.04	867.13	2772.55	1024.31	2757.06	1040.30	3331.61	1101.71	3308.76	1061.20	2983.59	1134.92
		S-SAD	2966.68	832.13	3087.03	922.16	3284.85	1286.09	3924.00	1227.60	3895.70	799.17	3164.22	839.18
		C-SAD	3199.63	1078.05	3167.17	865.07	3468.08	1494.41	3479.77	1083.26	3230.56	1149.78	3329.31	1163.53
		Hemispheric Assymetry (RT ms.)												
Condition	Visual field													
Verbal	Right	Control	541.73	126.41	562.48	96.59	565.93	101.01	550.38	97.30	546.42	72.48	554.52	113.29
		S-SAD	595.06	127.19	593.07	116.40	637.48	112.30	619.50	137.73	609.46	135.21	607.59	143.69
		C-SAD	568.93	112.52	608.13	123.40	616.46	119.56	588.49	116.29	571.47	100.09	579.44	124.72
	Left	Control	555.34	120.98	565.87	96.78	570.31	109.51	555.23	91.34	549.80	65.66	554.47	115.05
		S-SAD	588.29	117.62	591.01	104.38	642.33	113.50	619.44	135.15	608.29	123.85	603.89	129.38
		C-SAD	586.38	101.67	608.77	130.50	623.22	117.29	605.00	123.70	569.21	102.37	585.16	120.11
Spatial	Right	Control	557.69	110.92	584.45	116.91	569.23	97.38	565.42	99.77	573.11	81.29	575.61	141.07
		S-SAD	629.84	151.57	643.29	181.40	685.85	116.96	658.43	142.44	645.81	167.53	639.62	174.66
		C-SAD	611.77	156.41	689.51	196.56	661.50	149.62	603.64	134.88	587.21	128.34	591.92	136.19
	Left	Control	567.67	114.65	589.01	104.96	580.45	101.93	568.58	100.37	565.42	89.69	566.04	107.21
		S-SAD	641.81	152.88	651.73	173.55	690.38	129.77	646.42	130.26	658.43	144.18	650.61	191.69
		C-SAD	591.19	145.39	706.29	186.71	673.15	155.29	604.00	129.10	603.64	131.30	600.92	149.74

Note. $n = 21$ Control, 19 Subsyndromal SAD (S-SAD), and 17 Clinical (C-SAD) participants.

Digit Span. For Digit Span, trend analyses using planned polynomial contrasts and 6 x 3 (Month x Group) repeated measures MANOVAs were performed for the Forward and Backward conditions. No significant main effects or interactions were revealed from either the polynomial contrasts or the repeated measures MANOVAs for Forward or Backward Digit Span.

Visual Memory Span. For Visual Memory Span, planned polynomial contrasts were performed for the Forward and Backward conditions. There were no significant contrasts revealed for either Forward or Backward Visual Memory Span.

A 6 x 3 (Month x Group) repeated measures MANOVA was also conducted for the Forward and Backward conditions of Visual Memory Span separately, revealing main effects for Group in each condition. The group main effects for the Forward condition, $F(2,54) = 7.91, p = .001$, and the Backward condition, $F(2,54) = 10.54, p < .001$, indicated significant differences between the groups regardless of month. Subsequent post hoc analyses for the Forward condition showed controls to recall significantly more items than C-SADs and S-SADs. For the Backward condition significant differences were revealed between all pair-wise comparisons. Controls recalled significantly more items than C-SADs and S-SADs. S-SADs also recalled significantly more items than C-SADs.

Mental Rotation. For Mental Rotation, planned polynomial contrasts were performed revealing significant quadratic and cubic effects for month. The significant quadratic, $F(1, 51) = 8.07, p = .006$, and cubic effects for month, $F(1,$

51) = 6.15, $p = .016$, indicated a significant seasonal trend across the months. Figure 35 shows this seasonal pattern reflecting the slowest response time in August.

A 6 x 3 (Month x Group) repeated measures MANOVA was also performed with reaction time as dependent variable, revealing a significant main effect for Month. The main effect for month, Pillai's Trace = 0.25, $F(5, 47) = 3.18$, $p = .015$, indicated significant differences across the months regardless of group. Subsequent LSD post hoc pair-wise comparisons revealed a significantly slower reaction time for August than for all other months ($p < .05$) except October.

Hemispheric Asymmetry. For Hemispheric Asymmetry, planned polynomial contrasts were performed separately for left visual field (LVF), and for right visual field (RVF), in each condition (verbal and spatial). Overall, for the verbal condition, a significant cubic main effect was revealed for the RVF, although the LVF showed no significant quadratic or cubic effects. For the spatial condition, the RVF revealed a significant cubic main effect for month. For the LVF, significant quadratic and cubic main effects for month, and a significant cubic interaction between month and group, were revealed.

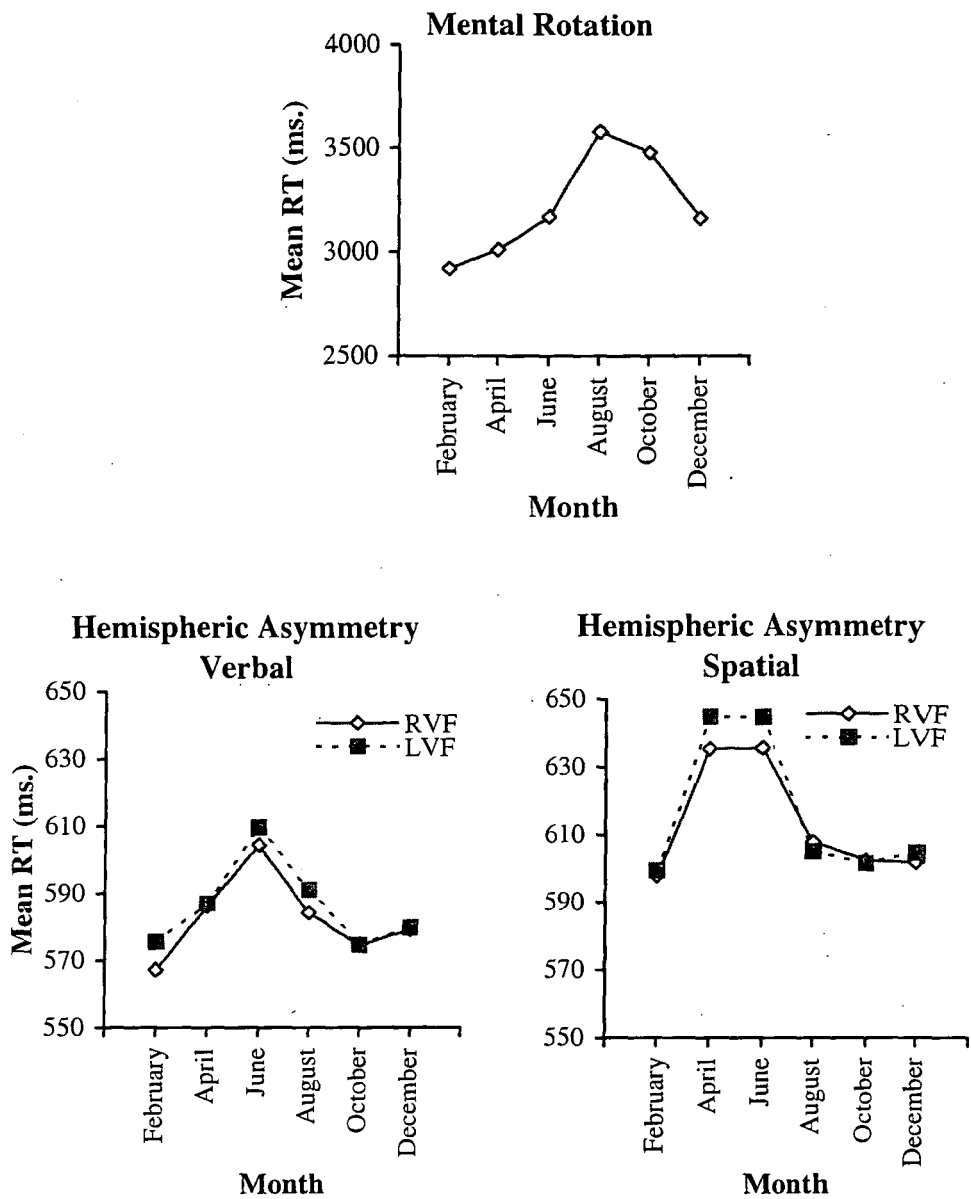


Figure 35. Seasonal pattern for participants overall for reaction time tasks: Mental Rotation, and verbal and spatial conditions of Hemispheric Asymmetry for participants overall at bimonthly sessions

For the verbal RVF condition, the significant cubic main effect for Month, $F(1, 53) = 5.17, p = .027$, indicated the reaction time varies across the months. Figure 9 reflects this cubic effect showing the main peak in reaction time for April and June.

For the spatial RVF condition, planned polynomial contrasts revealed a significant cubic main effect, $F(1, 54) = 5.22, p = .026$, which indicated that reaction time varied across the months for the right visual field. For the spatial LVF condition, planned polynomial contrasts revealed significant quadratic, $F(1, 54) = 5.35, p = .025$, and cubic, $F(1, 54) = 9.74, p = .003$ main effects, which indicated that the reaction time varied across the months for the left visual field. Planned polynomial contrasts also revealed a significant cubic interaction, $F(2, 54) = 3.46, p = .039$, indicating the variation across the months differed between the groups. Subsequent polynomial contrasts for each group separately revealed significant quadratic, $F(1, 16) = 5.17, p = .037$, and cubic, $F(1, 16) = 7.88, p = .013$, main effects for C-SADs only. Figure 36 shows C-SADs had the slowest reaction time in April and June, with a small secondary peak in December. This effect is not evident for control and S-SAD groups. Figure 36 reflects the same seasonal variations across the months for both the verbal and spatial conditions of the Hemispheric Asymmetry task. The verbal RVF condition, and both visual fields in the spatial condition show peak (slowest) reaction time in the autumn and winter months.

Multivariate $6 \times 2 \times 3$ (Month \times Visual Field: left/ right \times Group) repeated measures MANOVAs were also performed separately for each condition (verbal and spatial). Overall, for the verbal condition, MANOVA revealed no significant main effects of interactions. For the spatial condition, MANOVA revealed a significant main effect for month.

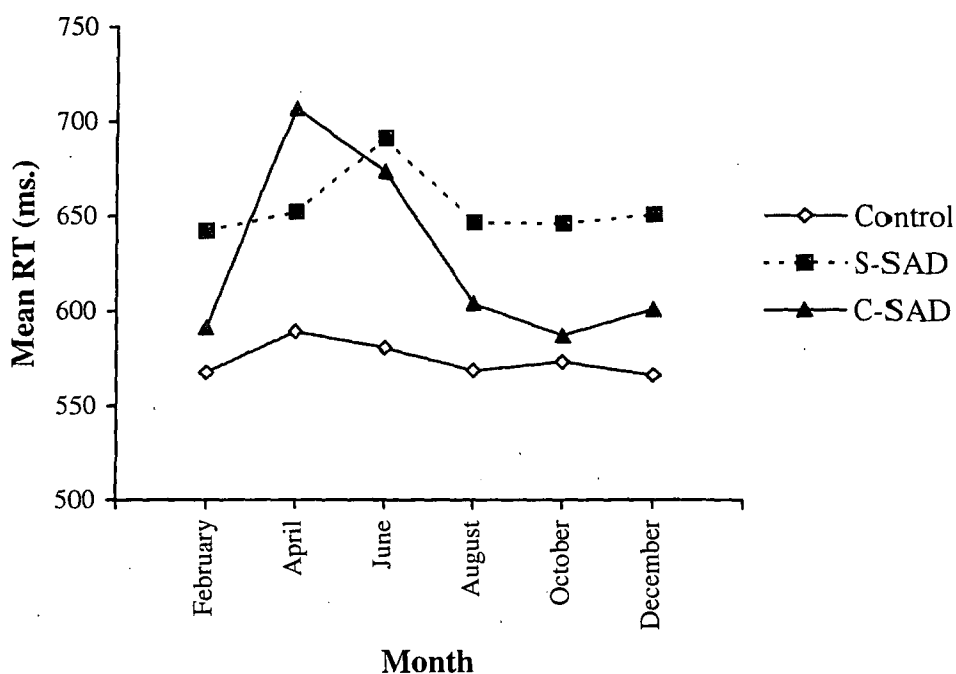


Figure 36. Seasonality shown for C-SADs only for the left visual field Spatial condition of Hemispheric Asymmetry

For the spatial condition, the main effect for month, Pillai's Trace = .25, $F(5, 50) = 3.33$, $p = .011$, indicated that reaction time varied across the months, regardless of group or visual field. Post hoc analyses showed significantly slower reaction times for April, and for June, than for all other months except August. The reaction time for June was also significantly longer than for August at the $p = .001$ significance level.

Summary of cognitive performance results. The Visual Memory Span task showed impairments in the number of items recalled for both SAD groups. Whilst the two SAD groups did not differ from each other in the Forward condition, C-SADs showed a greater impairment than S-SADs for the Backward condition. For the Mental Rotation task, seasonality, as indexed by significant quadratic and cubic

curves, was shown for all groups with a peak (slowest) reaction time in August. For the Hemispheric Asymmetry task, differential effects were found between verbal and spatial conditions, and between left and right visual fields. Seasonality, as indexed by significant quadratic and cubic curves, was shown for the RVF for all groups in both verbal and spatial conditions. For the spatial LVF condition, C-SADs only shows this impairment for April and June.

Emotion analyses

Mean self-rated scores and standard deviations for trait felt tension stress and effort stress over the past 30 days, due to four sources (work, family, finance, and own body) are shown in Tables 32 and 33 respectively.

The TESI was analysed in three parts: Part A, tension stress; Part B, effort stress; and Part C, the range of somatic and transactional emotions. Initial analyses used for Part A and Part B were trend analyses using planned polynomial contrasts which were conducted for each of the four stress sources (work, family, finance, and own body) for tension stress, and for effort stress, separately. Univariate repeated measures $6 \times 4 \times 3$ (Month \times Stress Source: work/family/finance/own body \times Group) ANOVAs were also conducted for tension stress (Part A), and for effort stress (Part B), separately.

Overall for tension stress from work, trend analyses revealed a significant quadratic main effect for month, and a significant quadratic interaction between month and group. For tension stress from own body, a cubic interaction between month and group was revealed.

Table 32

Means and Standard Deviations for Tension Stress Felt for Four Stressors (Work, Family, Finance, and Body) from the Tension and Effort Stress Inventory for Control, Subsyndromal SAD, and Clinical SAD Groups in the Thirty Days Prior to each of Six Bimonthly Testing Sessions

Source	Group	February		April		June		August		October		December	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Work	Control	2.95	1.94	3.95	1.80	4.24	2.12	3.81	2.06	3.90	1.73	3.43	2.04
	S-SAD	4.50	1.79	5.00	1.14	5.00	1.94	3.83	1.86	4.39	1.54	4.44	1.85
	C-SAD	3.12	1.80	4.88	1.36	4.94	1.95	4.71	1.65	5.35	1.66	3.12	1.93
Family	Control	2.43	1.50	2.43	2.01	2.38	1.69	2.33	1.65	2.57	1.89	2.14	1.31
	S-SAD	3.83	1.76	3.94	1.43	3.56	1.46	3.67	1.50	3.50	1.34	3.50	1.47
	C-SAD	3.24	1.56	3.29	1.40	3.53	1.66	3.47	1.62	3.29	1.57	4.29	1.72
Finance	Control	3.43	1.60	3.29	1.82	3.19	2.16	2.90	1.92	2.95	1.60	2.86	1.74
	S-SAD	3.67	1.61	3.67	1.57	3.72	1.93	3.72	1.81	3.78	1.90	3.44	1.82
	C-SAD	4.29	1.79	4.24	1.71	3.53	1.94	4.35	1.90	3.88	1.96	4.76	1.92
Body	Control	2.67	1.80	2.38	1.50	2.24	1.51	2.10	1.18	2.14	1.31	2.43	1.47
	S-SAD	4.06	1.16	3.89	1.45	3.83	1.72	4.28	1.67	3.83	1.20	3.78	1.31
	C-SAD	3.29	2.14	3.94	1.82	4.00	2.03	3.76	1.56	3.35	2.00	4.00	1.97

Note. Tension stress ratings were made on a 7-point scale (1 = not at all, 7 = very much)

n = 21 Control, 19 Subsyndromal SAD (S-SAD), and 17 Clinical SAD (C-SAD) participants.

Table 33

Means and Standard Deviations for Effort Stress Put up for Four Stressors (Work, Family, Finance, and Body) from the Tension and Effort Stress Inventory for Control, Subsyndromal SAD, and Clinical SAD Groups in the Thirty Days Prior to each of Six Bimonthly Testing Sessions

Source	Group	February		April		June		August		October		December	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Work	Control	3.48	1.91	4.14	1.71	4.14	2.01	3.24	1.89	4.10	1.76	3.38	2.27
	S-SAD	5.00	2.00	5.00	1.53	4.68	2.03	4.37	1.30	4.32	1.45	4.68	1.63
	C-SAD	3.76	2.14	4.82	1.42	4.71	2.17	4.47	1.70	5.24	1.56	3.41	2.03
Family	Control	2.48	1.54	1.90	1.34	2.33	1.91	2.05	1.40	2.43	1.57	2.48	1.47
	S-SAD	4.11	1.56	4.47	1.26	3.58	1.43	4.47	1.26	4.00	1.41	4.00	1.67
	C-SAD	3.71	1.93	3.59	1.73	3.59	1.50	3.24	1.35	3.59	1.46	4.29	1.90
Finance	Control	3.52	1.63	2.67	1.74	2.43	1.69	2.57	1.80	2.38	1.32	3.00	1.87
	S-SAD	3.68	1.60	3.74	1.82	3.26	1.52	4.37	1.64	3.26	1.48	3.63	2.03
	C-SAD	4.06	1.82	3.88	1.83	3.47	1.97	4.12	1.45	3.59	1.87	3.76	1.95
Body	Control	2.33	1.62	2.29	1.55	1.95	1.80	1.95	1.32	1.95	1.32	2.43	1.80
	S-SAD	4.05	1.43	3.58	1.54	3.26	1.56	4.00	2.03	3.95	1.47	3.89	1.73
	C-SAD	3.65	2.12	3.35	1.97	3.18	2.21	3.82	1.47	3.94	1.95	4.06	1.85

Note. Effort stress ratings were made on a 7-point scale (1 = not at all, 7 = very much)

n = 21 Control, 19 Subsyndromal SAD (S-SAD), and 17 Clinical SAD (C-SAD) participants.

For tension stress, the significant quadratic effect for work for month, $F(1, 54) = 14.54, p < .001$, and the quadratic interaction between month and group, $F(2, 54) = 15.30, p = .02$, indicated an overall seasonality, though the groups differ in their seasonal pattern for tension stress from work. Subsequent polynomial contrasts for each group separately revealed a significant quadratic effect for C-SADs only, $F(1, 16) = 15.22, p = .001$. From Figure 37, greater tension stress due to work is evident for C-SADs for the months from April to October, than for February and December.

Tension stress also showed a significant cubic interaction for own body between month and group, $F(2, 53) = 3.24, p = .03$, which indicated the groups differ in their seasonal pattern for tension stress from own body. Subsequent polynomial contrasts for each group separately also revealed a significant cubic effect for C-SADs, $F(1, 16) = 5.78, p = .029$ only. For own body (Figure 38), C-SADs show greater stress from their own body for April, June, and August, with a secondary peak evident for December.

Univariate analyses were also conducted for tension stress. For tension stress, significant main effects were revealed for stress source, for group, and a significant two-way Month x Stress Source interaction, and a significant three-way Month x Stress Source x Group interaction.

From the ANOVA for tension stress, the main effect for stress source, $F(2.77, 146.98) = 1.25, p < .001$, indicated significant differences in ratings between sources of stress in each stress type. The main effect for group, $F(2, 53) = 11.02, p < .001$, indicated significant differences between the groups. Subsequent post hoc

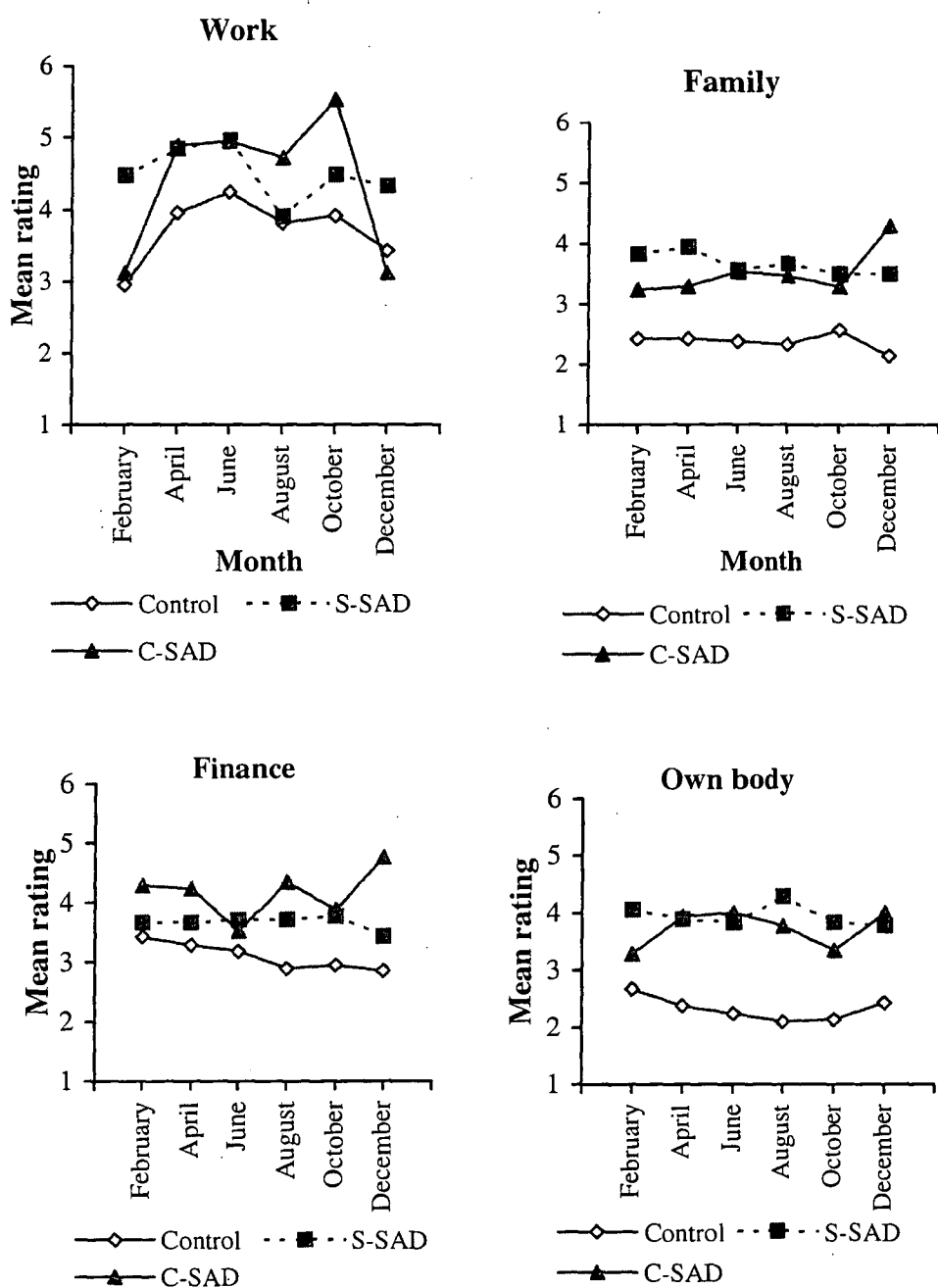


Figure 37. Tension Stress seasonality shown by Control, Subsyndromal SAD (S-SAD) and Clinical SAD (C-SAD) groups exposed to for work and own body in the thirty days prior to each of six bimonthly sessions

pairwise comparisons showed C-SADs and S-SADs to rate tension stress significantly higher than controls at the $p = .001$ significance level (Table 32). The two SAD groups did not differ from each other.

The significant Month x Stress Source interaction for tension stress, $F(9.38, 96.97) = 2.93, p = .002$, indicated ratings varied across month between the Stress Sources. The significant three-way Month x Stress Source x Group interaction, $F(18.75, 496.97) = 1.72, p = .03$, indicated that the groups varied in their ratings across the months for each stress source. Figure 37 shows the differential ratings in each source of stress. Univariate analyses were conducted for each group separately across the months revealing significant differences in tension stress for C-SADs for work only. Post hoc pair-wise comparisons showed C-SADs to rate work stress significantly higher for April, June, August, and October, than for February, at the $p = .01$ significance level. Univariate analyses for each month between the groups in each source of stress were also conducted. For work tension stress, controls rated significantly lower than S-SADs in February, and significantly lower than C-SADs in October. For family, significant differences were revealed in all months except October. During June, August, and December, the two SAD groups rated tension stress from family greater than the controls. For February and April, S-SADs only rated tension stress greater than the controls. For finance, differences were revealed in December only with C-SADs rating tension stress due to finance greater than S-SADs and controls. For own body, significant differences were revealed for all months. For February, S-SADs rated tension stress due to own body greater than controls, while for all other months both SAD groups rated tension stress due to own body greater than controls.

For effort stress from own body, trend analyses revealed a quadratic main effect from own body only. For effort stress, the significant quadratic effect for own body for month, $F(1, 54) = 4.36, p = .042$, indicated seasonality for participants overall regardless of group, for effort due to own body. Figure 38 shows participants to put less effort towards own body for April and June than all other months.

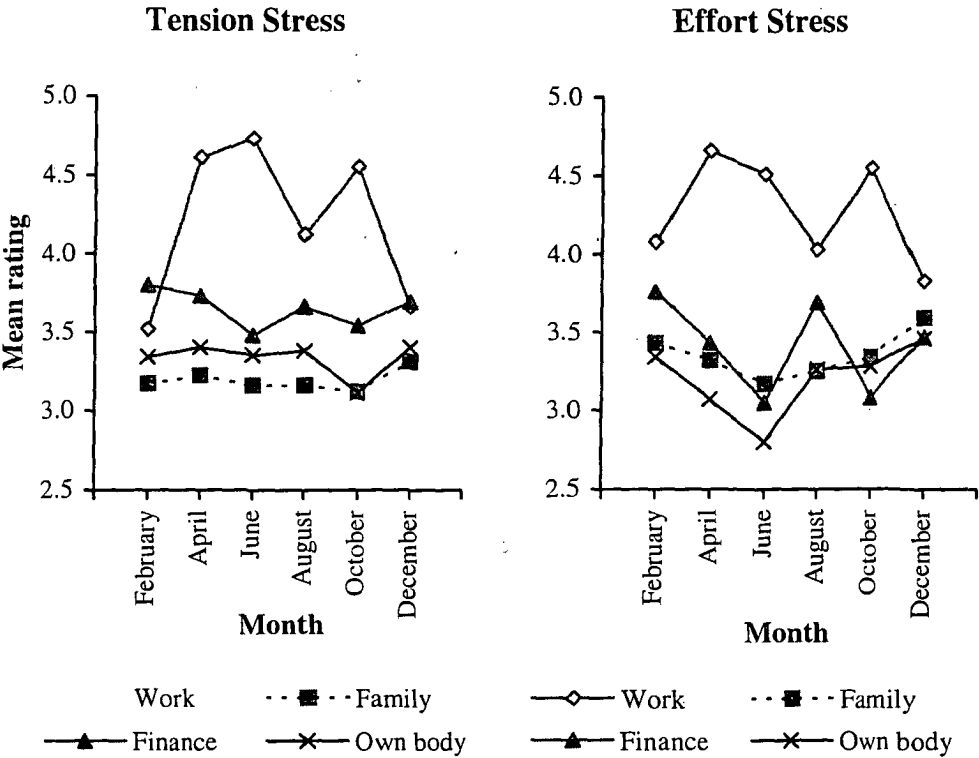


Figure 38. Degree of stress exposed to, and effort put up for four sources (work, family, finance, and body) by participants in the thirty days prior to each of six bimonthly testing sessions.

Univariate analyses were also conducted for effort stress. For effort stress, ANOVA revealed significant main effects for stress source, and for group, and a significant two-way Month x Stress Source interaction. The ANOVA for effort

stress revealed main effects for stress source, $F(2.70, 145.89) = 13.17, p < .001$, and group, $F(2, 54) = 17.27, p < .001$. The main effect for group indicated significant differences between the groups. Subsequent post hoc pair-wise comparisons showed C-SADs and S-SADs to rate effort stress significantly higher than controls at the $\alpha = .001$ significance level. The two SAD groups did not differ from each other.

The significant interaction between month and stress source for effort stress, $F(9.96, 538.03) = 2.72, p = .003$, indicated ratings varied across month between the stress sources. Subsequent univariate analyses for each month between sources of stress showed significant differences for all months except February and December. Post hoc pair-wise comparisons showed significantly greater effort stress was due to work than to family, finance, or own body in April, June, and October at the $p = .001$ significance level. For August, more effort was due to work than to family or body at the $p = .01$ significance level. Univariate analyses were also conducted for each source of stress across the months revealing significant differences for finance only. Post hoc pair-wise comparisons showed less effort for finance during June and October than during February and August.

For the final section of the Trait TESI (Part C), means and standard deviations for eight somatic emotions experienced in the thirty days prior to each bimonthly testing session are presented in two tables. Table 34 contains the four positive somatic emotions, and Table 35 contains the four negative somatic emotions.

Table 34

Means and Standard Deviations for Positive Somatic Emotions from the Tension and Effort Stress Inventory Experienced in the Thirty Days Prior to each of Six Bimonthly Sessions by Control, Subsyndromal SAD, and Clinical SAD Groups

Emotion	Group	February		April		June		August		October		December	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Relaxation	Control	4.57	1.6	4.19	1.40	4.14	1.56	1.26	1.26	4.24	1.37	4.81	1.44
	S-SAD	4.00	1.20	3.32	1.34	3.11	1.45	1.20	1.20	4.05	1.51	4.21	1.51
	C-SAD	3.76	1.68	3.29	1.21	4.00	1.58	1.22	1.22	3.82	1.47	4.24	1.6
Excitement	Control	4.90	1.34	4.67	1.62	4.67	1.43	1.46	1.46	4.76	1.41	4.86	1.24
	S-SAD	4.37	1.50	4.21	1.58	4.37	1.42	1.58	1.58	4.21	1.55	4.32	1.57
	C-SAD	4.53	1.42	3.12	1.27	3.65	1.50	1.32	1.32	4.12	1.69	4.41	1.77
Placidity	Control	3.57	1.43	3.33	1.53	3.24	1.09	1.12	1.12	3.00	1.10	3.33	1.46
	S-SAD	2.84	1.26	2.89	1.52	3.26	1.66	1.30	1.30	2.74	1.56	3.42	1.57
	C-SAD	3.18	1.55	3.24	1.39	3.29	1.53	1.05	1.05	2.94	1.34	3.29	1.4
Provocative-ness	Control	2.48	1.44	2.71	1.59	2.57	1.36	1.36	1.36	2.90	1.37	2.76	1.41
	S-SAD	3.21	1.32	3.68	1.95	3.32	1.63	1.16	1.16	3.32	1.49	3.37	1.46
	C-SAD	2.41	1.42	2.65	1.32	3.00	1.66	1.53	1.53	3.00	1.41	2.94	1.68

Note. $n = 21$ control, 19 subsyndromal SAD (S-SAD), and 18 Clinical SAD (C-SAD).

Table 35

Means and Standard Deviations for Negative Somatic Emotions from the Tension and Effort Stress Inventory Experienced in the Thirty Days Prior to each of Six Bimonthly Sessions by Control, Subsyndromal SAD, and Clinical SAD Groups

Emotion	Group	February		April		June		August		October		December	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Anxiety	Control	3.00	1.87	3.57	1.86	4.14	1.77	3.48	1.86	3.71	1.74	3.24	1.67
	S-SAD	3.37	1.80	4.32	1.67	4.47	1.68	3.84	1.38	3.95	1.58	3.74	1.73
	C-SAD	3.82	1.67	5.24	1.25	5.59	1.00	4.71	1.96	4.71	1.86	3.76	1.86
Boredom	Control	2.95	1.99	2.57	1.60	2.90	1.58	2.14	0.91	2.38	1.28	2.57	1.5
	S-SAD	2.21	1.58	2.26	1.19	2.42	1.80	2.53	1.68	2.26	1.56	2.11	1.29
	C-SAD	2.76	1.82	3.06	1.56	3.76	2.11	3.41	1.66	2.71	1.79	2.88	2.06
Anger	Control	2.57	1.54	2.48	1.47	2.52	1.60	2.48	1.47	2.62	1.47	2.62	1.32
	S-SAD	2.89	1.24	3.32	1.86	3.68	1.70	3.58	1.43	3.32	1.70	3.16	1.8
	C-SAD	3.06	1.78	4.12	1.73	3.35	1.66	3.12	1.58	3.47	1.97	3.59	1.46
Sullenness	Control	1.95	1.20	2.14	1.35	2.48	1.21	2.24	1.30	2.57	1.60	2.48	1.44
	S-SAD	1.89	0.88	2.42	1.22	2.63	1.30	2.16	1.01	2.16	1.30	2.26	1.19
	C-SAD	2.18	1.59	3.12	1.54	2.88	1.83	2.76	1.44	2.41	1.33	2.47	1.59

Note . n = 21 control, 19 subsyndromal SAD (S-SAD), and 18 Clinical SAD (C-SAD).

Somatic Emotions. Individual trend analyses using planned polynomial contrasts conducted for each of the eight somatic emotions separately revealing significant quadratic effects for month for relaxation, anxiety, and sullenness, and cubic main effects for month for anxiety, and sullenness. Trend analyses showed significant quadratic effects for month for relaxation, $F(1, 54) = 6.16, p = .016$, anxiety, $F(1, 54) = 32.75, p < .001$, and sullenness, $F(1, 54) = 4.22, p = .045$. Significant cubic effects were also revealed for anxiety, $F(1, 54) = 7.43, p = .009$, and sullenness, $F(1, 54) = 5.63, p = .021$. From Figure 39, relaxation shows a trough, and anxiety and sullenness show a peak during April and June. There were no significant seasonal effects for excitement, placidity, provocativeness, boredom or anger.

Univariate $6 \times 4 \times 3$ (Month \times Emotion \times Group) repeated measures ANOVAs were also conducted for each somatic category separately: positive somatic emotions (excitation, relaxation, placidity, and provocativeness), and negative somatic emotions (boredom, anxiety, anger, and sullenness).

Univariate analyses conducted for positive somatic emotions revealed significant interactions between month and emotion, and between emotion and group. For the negative somatic emotions, ANOVA revealed main effects for month and for group, and significant interactions between month and emotion, and between emotion and group.

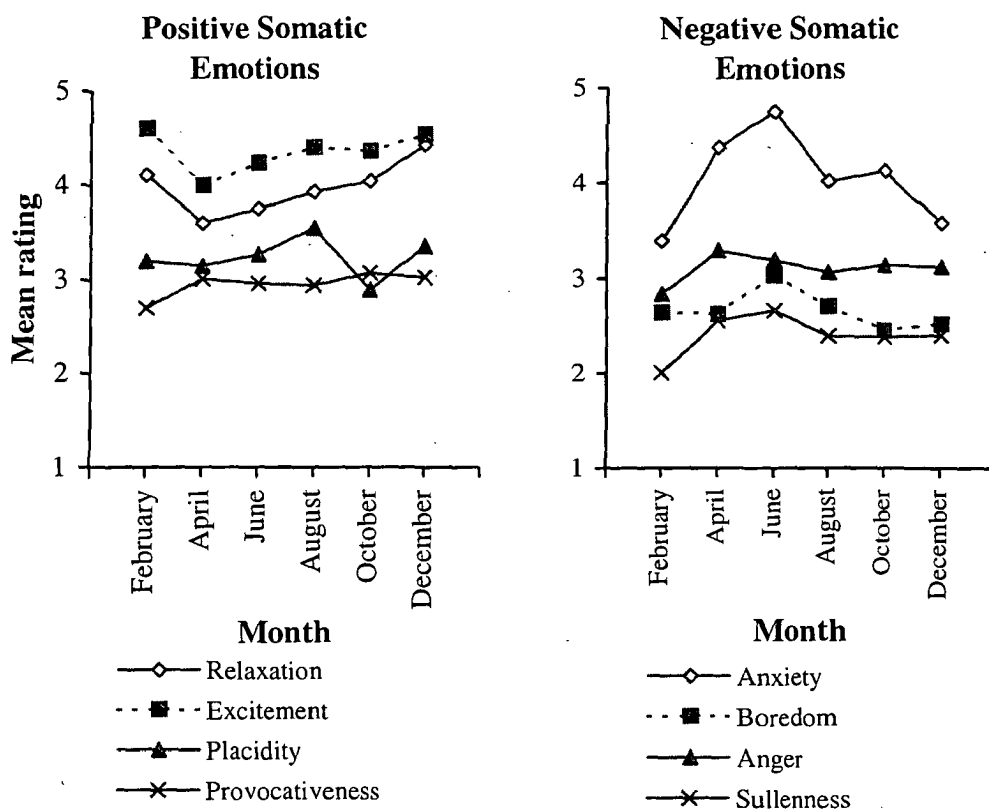


Figure 39. Participant ratings for positive (relaxation, excitement, placidity, and provocativeness) and negative (anxiety, boredom, anger, and sullenness) somatic emotions experienced in the thirty days prior to each of six bimonthly testing sessions.

For the positive emotions, the interaction between month and emotion, $F(10.14, 547.39) = 2.05, p = .026$, indicated that ratings for positive emotions varied across the months. Univariate repeated measures analyses were conducted across the months for each emotion separately which revealed significant differences for relaxation only. Post hoc pair-wise comparisons indicated participants felt less relaxed in April than in February, October, and December. Participants were also less relaxed in June and August than in December. The interaction between emotion and group, $F(5.09, 547.39) = 2.67, p = .017$, as shown in Figure 40 indicated that

the groups varied in their ratings between the emotions. Univariate analyses were conducted between the groups for each emotion that revealed no significant group differences.

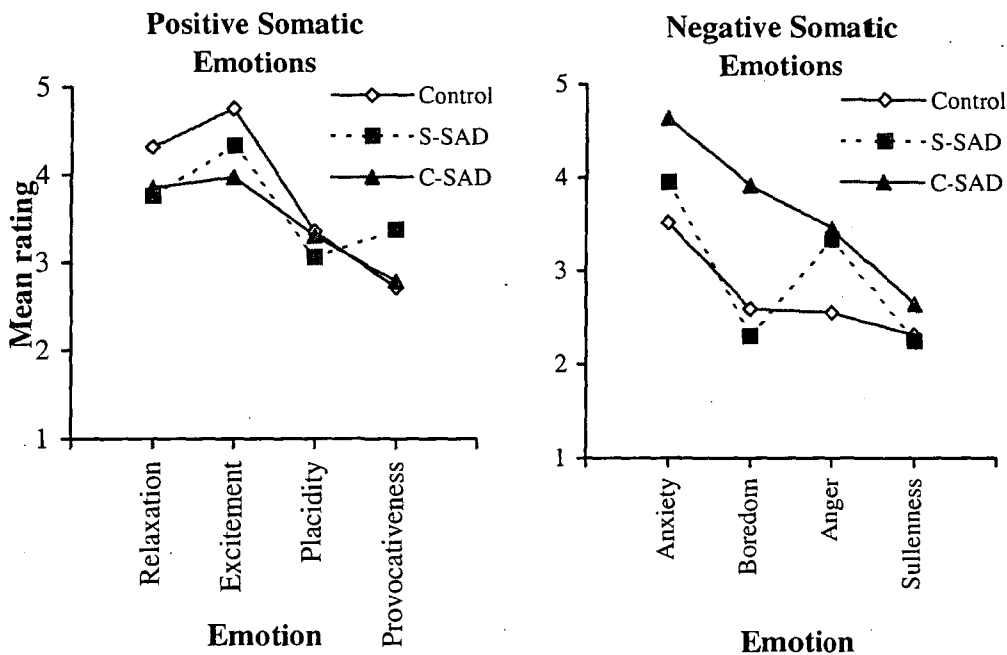


Figure 40. Positive (relaxation, excitement, placidity, and provocativeness) and negative somatic emotion (anxiety, boredom, anger, and sullenness) ratings for control, subsyndromal SAD (S-SAD), and clinical SAD (C-SAD) groups

For the negative emotions, main effects were revealed for month, $F(4.42, 227.75) = 4.31, p = .002$, and for group, $F(2, 54) = 3.39, p = .041$. The interaction between month and emotion, $F(5.49, 590.91) = 2.27, p = .026$, indicated the ratings for emotion varied across the months in each category. Subsequent repeated measures ANOVAs across the months for each emotion separately revealed significant differences for anxiety, and sullenness. Participants felt more anxiety in June than in all other months except April. Participants were also more sullen in April, June, and August than in February. Figure 39 shows higher ratings for

anxiety and sullenness, during the autumn/ winter months. For the negative emotions, the interaction between emotion and group, $F(5.49, 590.91) = 2.48, p = .03$, indicated that the groups varied in their ratings for the emotions. Univariate analyses were conducted between the groups for each emotion revealing significant differences for anxiety, boredom, and anger. C-SADs felt more anxiety and anger than controls. S-SADs also felt more anger than controls. C-SADs felt more boredom than S-SADs. Figure 40 shows these group differences for anxiety, boredom, and anger.

Also in the final section of the Trait TESI (Part C), means and standard deviations for eight transactional emotions experienced in the thirty days prior to each bimonthly testing session are presented in two tables. Table 36 contains the four positive transactional emotions, and Table 37 contains the four negative transactional emotions.

Transactional Emotions. Individual trend analyses using planned polynomial contrasts conducted for each of the eight transactional emotions separately revealing significant cubic effects for month for pride, virtue, shame, and guilt, and a significant interaction between month and group for virtue. Strong tendencies were also revealed for humiliation to show a cubic main effect, and for guilt to show a cubic interaction.

Trend analyses conducted on individual transactional emotions showed significant cubic effects for month for pride, $F(1, 54) = 4.88, p = .031$; virtue, $F(1, 54) = 4.89, p = .031$; shame, $F(1, 54) = 5.66, p = .021$; and guilt, $F(1, 54) = 6.42, p = .014$, indicating a seasonal pattern across the months for each of these emotions.

Table 36

Means and Standard Deviations for Positive Transactional Emotions from the Tension and Effort Stress Inventory Experienced in the Thirty Days Prior to each of Six Bimonthly Sessions by Control, Subsyndromal SAD, and Clinical SAD Groups

Emotion	Group	February		April		June		August		October		December	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Pride	Control	3.76	1.84	3.81	1.94	3.38	1.86	4.29	1.79	3.86	1.59	3.62	1.94
	S-SAD	3.68	1.53	3.68	1.53	2.89	1.66	4.00	1.45	3.95	1.18	4.21	1.51
	C-SAD	3.18	1.81	2.71	1.61	3.12	1.58	2.82	1.33	3.12	1.27	2.82	1.85
Modesty	Control	2.76	1.41	3.43	1.43	2.81	1.36	3.62	1.36	3.05	1.28	3.05	1.20
	S-SAD	3.79	1.23	3.63	1.50	3.47	1.35	3.63	1.01	3.58	1.17	3.47	1.35
	C-SAD	3.41	1.42	2.82	1.24	2.82	1.38	2.76	1.44	2.76	1.44	2.71	1.79
Gratitude	Control	4.38	1.77	4.62	1.36	4.67	1.35	4.86	1.24	4.24	1.51	4.00	1.45
	S-SAD	4.58	1.30	4.47	1.54	4.32	1.16	4.58	1.39	4.47	1.54	4.53	1.43
	C-SAD	4.53	1.97	3.82	1.38	3.65	1.06	3.82	1.63	3.88	1.65	3.41	1.97
Virtue	Control	2.95	1.56	3.43	1.54	3.24	1.51	3.00	1.30	3.14	1.39	3.00	1.41
	S-SAD	3.47	1.26	3.47	1.07	2.84	1.26	3.53	1.31	3.47	1.12	3.32	1.16
	C-SAD	3.24	1.82	2.41	0.94	2.88	1.50	2.82	1.29	3.41	0.71	2.41	1.18

Note. $n = 21$ control, 19 subsyndromal SAD (S-SAD), and 18 Clinical SAD (C-SAD).

Table 37

Means and Standard Deviations for Negative Transactional Emotions from the Tension and Effort Stress Inventory Experienced in the Thirty Days Prior to each of Six Bimonthly Testing Sessions by Control, Subsyndromal SAD, and Clinical SAD Groups

Emotion	Group	February		April		June		August		October		December	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Humiliation	Control	1.33	0.58	1.71	1.27	1.86	1.31	1.67	0.91	1.90	1.48	1.52	0.93
	S-SAD	2.05	1.08	2.26	1.45	2.21	1.27	2.26	1.37	2.05	1.31	2.32	1.45
	C-SAD	1.82	1.19	2.76	1.92	2.35	1.32	1.94	1.20	2.12	1.32	2.18	1.33
Shame	Control	1.43	0.93	1.57	1.25	1.62	1.36	1.29	0.56	1.86	1.53	1.62	1.28
	S-SAD	2.32	1.20	2.37	1.54	2.74	1.33	2.00	1.37	1.89	1.10	2.16	1.61
	C-SAD	2.00	1.66	2.88	1.80	2.18	1.47	1.82	1.29	1.94	1.34	2.00	1.22
Resentment	Control	2.24	1.34	1.81	1.36	2.57	1.78	2.00	1.41	2.76	1.79	2.38	1.69
	S-SAD	2.47	1.35	3.05	1.68	2.47	1.43	2.89	1.66	2.89	1.70	2.53	1.71
	C-SAD	2.24	1.64	2.94	1.71	2.94	1.68	2.59	1.62	3.24	1.68	3.47	1.70
Guilt	Control	1.52	0.93	1.57	1.36	1.86	1.42	1.48	0.81	2.24	1.87	1.81	1.17
	S-SAD	2.11	1.52	2.63	1.50	3.00	1.94	2.42	1.46	2.00	1.56	2.26	1.76
	C-SAD	2.53	1.81	3.41	1.87	3.00	1.87	2.24	1.39	2.65	1.73	2.71	1.61

Note . n = 21 control, 19 subsyndromal SAD (S-SAD), and 18 Clinical SAD (C-SAD).

The seasonal effects for significant transactional emotions are reflected in Figure 41, which shows a trough for the positive emotions pride and virtue, and a peak for the negative emotions shame and guilt, for the autumn and winter months.

The significant cubic interaction between Month and Group for virtue, $F(1,54) = 5.75, p = .005$, indicated the seasonal pattern varied between the groups. Follow up analyses on each group separately showed a significant effect for C-SADs, $F(5, 12) = 4.50, p = .015$, only. C-SADs show a trough for virtue for the autumn and winter months, and a secondary trough for December (Figure 43).

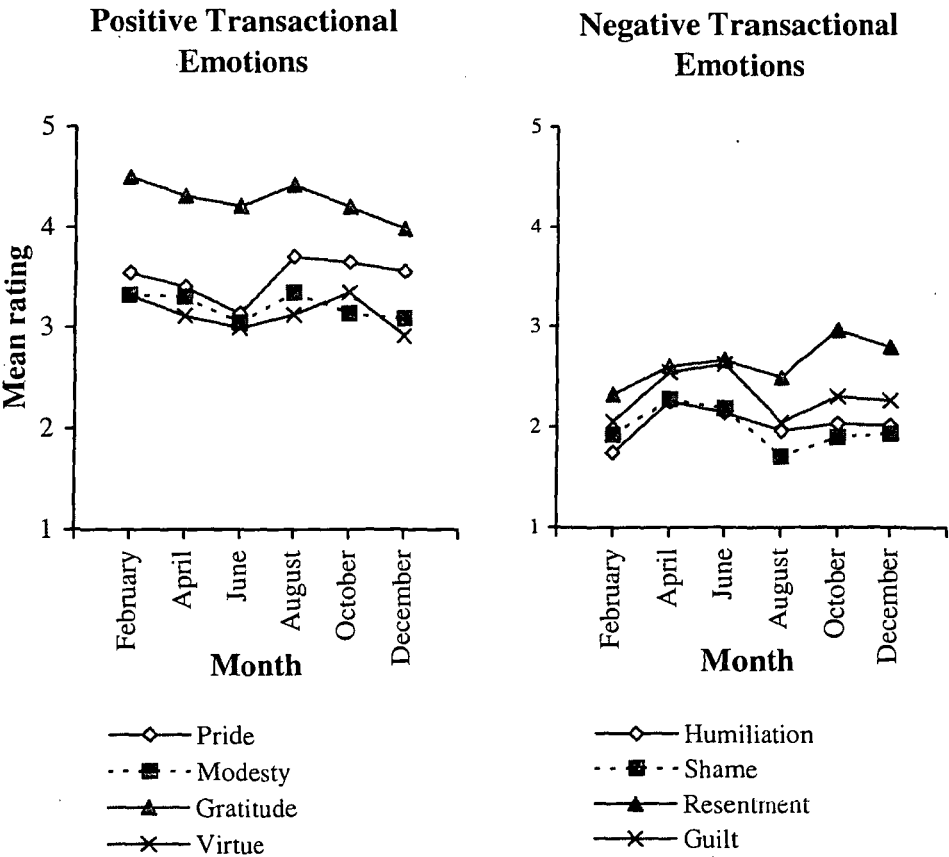


Figure 41. Seasonal pattern for transactional emotions from the Tension and Effort Stress Inventory shown by participants overall for pride, modesty, gratitude, virtue, humiliation, shame, resentment, and guilt.

Planned polynomial contrasts for the transactional emotions also revealed strong tendencies for humiliation to show a cubic main effect, $F(1, 54) = 3.90, p = .053$, and for guilt to show a cubic interaction, $F(2, 54) = 3.08, p = .054$. For guilt, subsequent polynomial contrasts were conducted for each group separately which showed the tendency to be due to significant cubic main effects for S-SADs, $F(1, 18) = 5.44, p = .031$, and for C-SADs, $F(1, 16) = 6.19, p = .024$. Controls showed no significance. From Figure 42, the C-SAD group show a peak for April, and a trough for August, and also show a secondary peak for October and December. Figure 42 also shows a peak for S-SADs for April and June, and a trough in October. Figure 42 also shows a secondary peak for December.

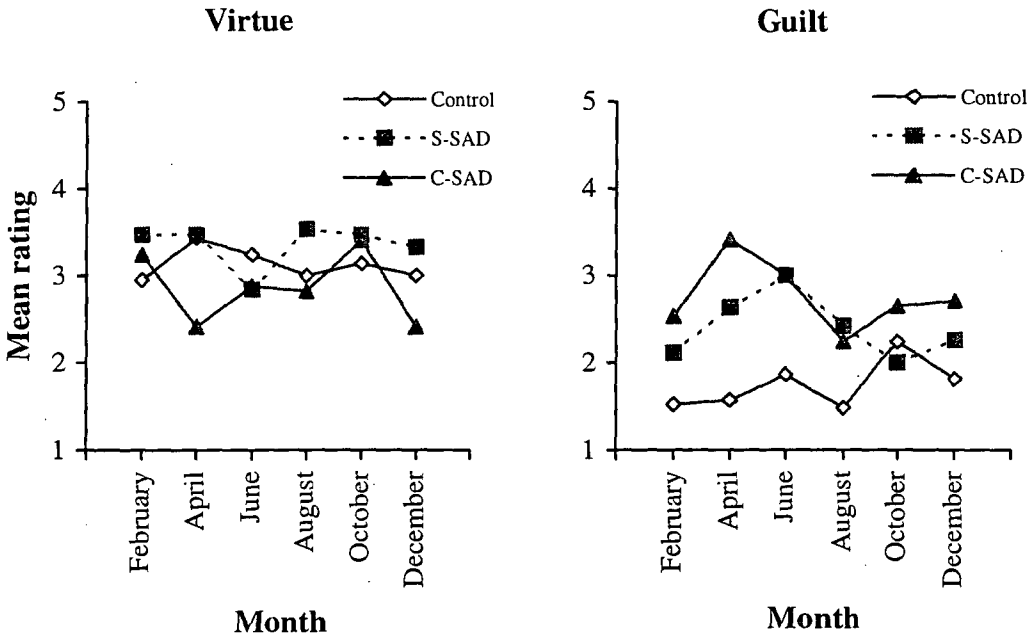


Figure 42. Seasonality for virtue and guilt reflecting differential seasonal pattern between the groups

Univariate analyses were also conducted for positive and negative transactional emotions, separately. For the positive transactional emotion category,

ANOVA revealed a significant interaction between Month and Group. For the negative transactional emotion category, ANOVA revealed a significant main effect for group. A strong tendency for a main effect for month was also revealed for the negative category.

For the positive emotions, the significant interaction between Month and Group for positive emotions, $F(8.32, 224.53) = 1.98, p = .048$, indicated the groups varied in their ratings across the months. Figure 43 shows these groups differences in ratings across the months. Subsequent univariate analyses were conducted between the groups for each month separately, with significant differences revealed for April, August, and December. Figure 43 shows that for April and August, C-SADs rated positive transactional emotions significantly lower than S-SADs and controls. For December, C-SADs rated significantly lower than controls. Repeated measures analyses were conducted across the months for each group separately, revealing significant differences for the S-SAD group only. S-SADs rated positive transactional significantly lower in June than in all other months.

For the negative emotions, the main effect group, $F(2, 54) = 3.39, p = .041$, indicated that ratings for negative transactional emotions vary between the groups. C-SADs and S-SADs rated the negative transactional emotions significantly higher than controls (see Table 37). The strong tendency for a main effect for month for negative emotions, $F(4.23, 228.43) = 2.35, p = .052$, was most likely due to the emotions being rated higher in April and June than in February, and in August.

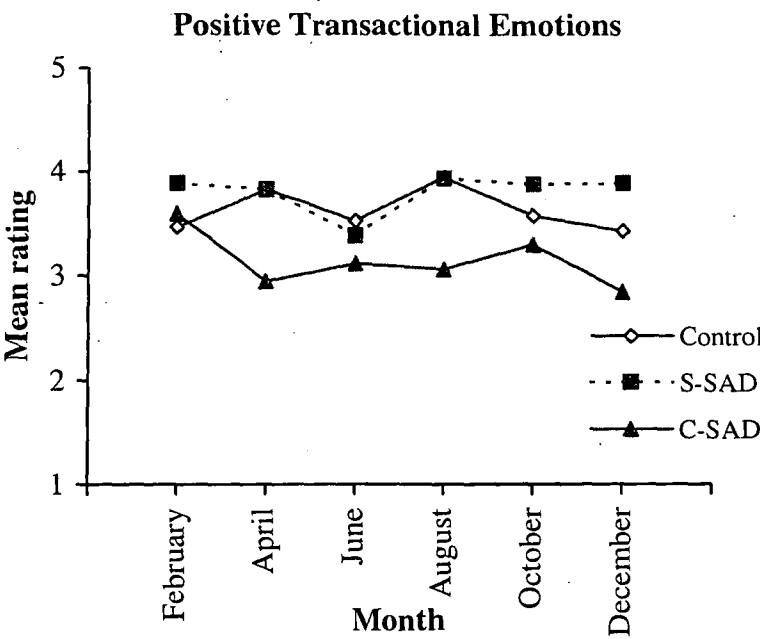


Figure 43. Positive transactional emotion ratings (pride, modesty, gratitude, and virtue) for control, subsyndromal SAD (S-SAD) and clinical SAD (C-SAD) groups experienced for six bimonthly testing sessions.

Current felt stress. Means and standard deviations for stress felt at ‘the present moment’ from the state version of the TESI for each of six bimonthly testing sessions are presented in Table 38.

Trend analyses using planned polynomial contrasts was conducted for current felt stress revealing a significant quadratic effect for month, $F(1, 53) = 5.36, p < .03$, indicating participants overall showed seasonality across the twelve month period, regardless of group. Figure 44 shows this seasonal effect with peaks for April and October and troughs for June and December.

Table 38
Means and Standard Deviations for Current Stress Ratings from the State Tension and Effort Stress Inventory for Control, Subsyndromal SAD, and Clinical SAD Groups at Bimonthly Testing Sessions

	February	April	June	August	October	December
Group	Means (SD)					
Control	2.33 (1.53)	2.48 (1.54)	2.00 (1.10)	2.38 (1.47)	2.71 (1.85)	1.90 (1.22)
S-SAD	3.17 (1.58)	3.39 (1.72)	3.06 (1.43)	3.17 (1.20)	3.39 (1.54)	2.61 (1.29)
C-SAD	3.24 (1.64)	4.12 (1.50)	3.53 (1.18)	3.59 (1.62)	3.41 (1.97)	3.12 (1.36)

Note. $n = 21$ Control, 19 Subsyndromal SAD (S-SAD), and 17 Clinical SAD (C-SAD) participants. Ratings were from 1 (no stress) to 7 (high stress)

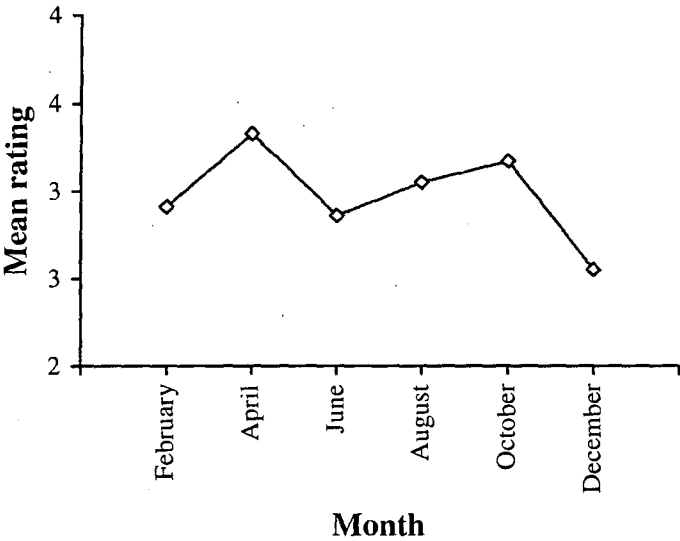


Figure 44. Current felt stress ratings from the State Tension and Effort Stress Inventory for participants at bimonthly sessions

A multivariate repeated measures 6 x 3 (Month x Group) MANOVA was also conducted for current stress, revealing main effects for Month, and for Group. The interaction was not significant.

The main effect for Month, $F(5, 49) = 3.58, p < .008$, indicated participants varied in their 'current felt' stress level across the Months. Post hoc pair-wise comparisons showed higher ratings for stress for April than for February, June, and December. The stress ratings were also higher in February, August, and October, than in December. The main effect for Group, $F(2, 53) = 5.27, p = .008$, indicated the groups varied in their 'current felt' stress ratings. Post hoc pair-wise comparisons showed C-SAD and S-SAD groups to rate their 'current felt' stress higher than controls. The two SAD groups did not differ from each other.

Summary of emotion analyses. The trait version of the TESI was analysed in 3 parts: For Parts A and B, seasonality was shown for C-SADs only reflecting greater tension stress from work for the months of the university semesters. C-SADs also feel greater tension stress from their own body for the autumn and winter months, though all participants put less effort towards their own body for April and June. A three-way interaction for tension stress was due to (a) C-SADs feeling greater tension stress from work for April, June, August, and October, than for February (b) both SAD groups feeling greater tension stress from family as compared to controls for June, August, and December and (c) both SAD groups feeling greater tension stress from own body for all months except February as compared to controls. For Part C, seasonality was shown for participants overall for the somatic emotions, relaxation, anxiety, and sullenness, and the transactional emotions, pride, shame, and guilt. Of these, the positive emotions showed a trough, and the negative emotions showed a peak, for April and June. C-SADs only felt less virtue for the autumn and winter months.

Current felt stress showed a peak for April and October for all participants. Overall, both SAD groups rated current felt stress greater than controls, though the SAD groups did not differ from each other.

Discussion

Screening questionnaire

The screening questionnaire: SPAQ, differentiated between the groups for all symptoms. The two SAD groups rated a greater degree of seasonal change to all symptoms as compared to controls. C-SADs also rated these seasonal changes greater than S-SADs for all symptoms except sleep. The SAD groups varied across the seasons in the number of hours slept, while controls did not differ. For winter, C-SADs slept longer hours than S-SADs and controls. For summer, S-SADs slept less than controls.

Depression and behavioural symptoms

For Longitudinal Study 2, the two SAD groups showed increases in depression and behavioural symptoms during winter as predicted. C-SADs and S-SADs showed increases in depression for the autumn and winter months evidenced by significant trend analyses for the two depression scales, the Beck Depression Inventory with addendum, and the WMI scale for depressed mood and ideation. Both SAD groups also showed increases in the atypical vegetative symptoms scale, and decreases for hypomania mood and ideation scale in winter. This seasonal pattern was not shown for the WMI scale for typical vegetative symptoms, or for the controls on any scale.

Thus the presence of depression and associated atypical vegetative symptoms during autumn and winter, and symptom remission and an increase in hypomania symptoms during spring and summer, validated the further administration of cognitive tasks. The WMI scale for typical vegetative symptoms showed random depressive episodes across the twelve-month period, demonstrating the non-seasonal nature of these symptoms.

Differential group effects were shown for the sleep quality scales. Regardless of month, C-SADs experienced worse nights, more disturbed nights, they slept lighter, and were more fatigued than controls. The study also showed changes to food preference across the twelve-month period. A greater percentage of C-SADs consumed fats and carbohydrates during winter. A greater percentage of controls also show a preference for carbohydrates in June as compared to all other months. This variation was not evident for protein intake. These changes to food preference are consistent with those found by Lacoste and Wirz-Justice (1989) in SAD participants.

Impairment to cognitive performance

The current study showed no differences for either forward or backward conditions of the verbal memory task, Digit Span, either between the groups or across the twelve-month period. This finding differs from the previous finding of a decreased short-term memory in winter, as compared to a non-winter period (Austen & Wilson, 1998). On the other hand, the current study showed group differences for the Visual Memory Span that varied between forward and backward conditions. For the forward condition, the SAD groups recalled significantly less correct items than the control group, though did not differ from each other. For the

backward condition, whilst both SAD groups recalled significantly less items than controls, C-SADs also recalled less correct items than S-SADs. Deficits to the forward condition implicated short-term memory impairment for both SAD groups. In contrast, the additional cognitive load from the backward condition or working memory dissociates between the two SAD groups and indicates greater impairment for C-SADs as compared to S-SADs. Findings for both visual memory and verbal memory were independent of time of year and overall they suggest impairment in the visual pathway that does not remit during the spring and summer months. The deficits for SADs are consistent with findings from Michalon et al. (1997) who showed that whilst mood and seasonal symptoms improved in summer, visual memory performance did not.

Hemispheric lateralisation

Seasonality for the Hemispheric Asymmetry task was evident from the trend analyses, though seasonality varied between the verbal and spatial conditions and hemispheres. For the verbal condition, there were no group differences, however, seasonality was shown for participants for the right visual field only. Participants' reaction time was slower for the left hemisphere during the autumn and winter months as compared to the spring and summer. This finding is consistent with the traditional view that verbal processing is lateralised to the left hemisphere with an impaired functioning for winter. An alternative explanation could be that if the verbal stimuli were processed in the right hemisphere as suggested by Volf et al. (1993) the longer reaction time in winter may be due to interference in processing with negative emotion that also occupies the right hemisphere.

For the spatial condition, seasonality was evident for both hemispheres for participants overall, reflecting a slower reaction time during the winter months. For the LVF, or the right hemisphere, this impairment was evident for C-SADs only, indicative of the dual processing interference between spatial processing and negative emotion and supporting previous research for a right hemispheric dysfunction in depression. Previous research (e.g., Allen et al., 1993; Volf et al., 1993) has suggested an asymmetrical hemispheric pattern specific to SAD that may implicate vulnerability. However findings have not been consistent and further research is required for confirmation that the finding for the current study is specific to SAD, and distinct from NSD. For the RVF, or left hemisphere, a seasonal effect was also evident for participants regardless of group for the spatial condition, with longer reaction times for winter. Any explanation of this result is not clear. It is possible that an additional response time relates to a transfer of information to the right hemisphere for processing. Alternatively, the spatial stimuli could have been processed as verbal stimuli in the left hemisphere, thus showing psychomotor retardation in the winter months. All participants were right handed, hence any effect from a non-dominant specialisation would be minimal.

For the mental rotation task participants showed a slower reaction time for August than for all other months regardless of group. The task was selected for its pure spatial properties, and hence right hemispheric processing, and may further implicate interference in the right hemisphere between cognitive processing and negative emotion. The slowed response time may also be due to greater level of fatigue in winter reported on the sleep quality scale “refreshed/ fatigued” for all participants regardless of group.

Emotion

For tension stress, the groups varied in their ratings between the four stressors across the months. Seasonality was shown for tension stress from work and own body for the C-SAD group only. C-SADs felt greater tension stress from work from April to October (university semester months) as compared to February and December (university summer vacation). C-SADs also felt greater tension stress from their own body in all autumn and winter months as compared to February and October. A smaller peak was evident for December. C-SADs felt greater financial tension stress as compared to S-SADs and controls in December. During June, August, and December, both SAD groups felt greater tension stress from family as compared to controls.

Effort stress ratings varied between stressors across the months, but did not differ between the groups. All participants reported less effort for own body in April and June.

For both the positive and negative somatic emotion categories, emotion interacted with month. Regardless of group, for the positive emotions participants were less relaxed for April and June. For the negative emotions, participants showed more anxiety and sullenness during the autumn and winter months. Emotion also interacted with group in both positive and negative somatic emotion categories. The interaction for the positive emotion category was due to differences between the emotions, with no group differences evident. In contrast, group differences were shown for the negative emotion category. C-SADs felt more anxiety and anger than controls, and more boredom than S-SADs. S-SADs also felt more anger than controls.

For the transactional emotions, C-SADs showed less virtue during the autumn and winter months, and both SAD groups showed more guilt in April and June. Regardless of emotion, C-SADs reported less positive emotion in April and August as compared to S-SADs and controls. C-SADs reported less positive emotions in December as compared to controls. Regardless of month or emotion, the two SAD groups rated negative transactional emotions higher than controls.

'Current felt' stress was also rated higher by both SAD groups as compared to controls, though the SAD groups did not differ from each other.

Seasonality in the general population

Seasonality for participants overall was shown for several variables. Regardless of group, participants were more fatigued in April and June as compared to all other months, and this seasonal variation to fatigue was greater for C-SADs than for controls. A tendency was shown for all groups to show a preference for carbohydrates during autumn and winter, and this tendency was greater for the SAD groups than for controls.

Seasonality was shown for cognitive efficiency with tasks showing impairment during the autumn and/ or winter months. All participants showed a slower response time for Mental Rotation in August. For the Hemispheric Asymmetry tasks, seasonality varied across verbal and spatial conditions, as well as left and right hemispheres. For both verbal and spatial conditions retardation in cognitive processing was shown for the left hemisphere only.

All participants expended less effort stress for own body during April and June. Several emotions showed seasonality across the twelve-month period. These were the somatic emotions: relaxation, anxiety, and sullenness, and the transactional emotions: pride, virtue, shame, and guilt. Of these, regardless of group, participants showed a trough for the positive emotions, and a peak for the negative emotions during winter. Participants felt less relaxed, and showed less pride and virtue during winter. Participants also felt more anxious, were more sullen, and showed more shame and guilt during the winter months. 'Current felt' stress ratings varied across the twelve-month period, with peaks shown in April and October, and troughs in June and December. The troughs most likely reflect a relief from stress at the completion of the university examination period.

Implications

Longitudinal Study 1 of autonomic arousal (Chapter 7) has shown mood and associated atypical vegetative behavioural symptoms specific to the autumn/ winter seasons, and their remission during the spring/ summer seasons. Although there is substantial overlap of participants, Longitudinal Study 2 of cognitive performance has shown similar findings, further confirming the seasonal nature of SAD. Depression, as indexed by the BDIadd and the depressed mood and ideation (DMI) scale from the WMI, was higher for the SAD groups as compared to controls and also showed peaks in autumn/ winter. The WMI scales for atypical vegetative symptoms (AVS) and hypomania mood and ideation (HMI) showed symptoms for the SAD groups specific to the autumn and winter months. The SAD groups showed higher AVS scores, and lower HMI scores in April, June, and August compared to controls, and also scores for spring and summer. Longitudinal Study 2

also showed additional evidence supporting the SAD symptoms. Differential group effects were shown for the sleep quality subscales. The C-SAD group experienced worse nights, slept lighter, and were more fatigued as compared to controls. C-SADs also had more disturbed nights than S-SADs and controls. Both SAD groups showed greater preferences for fats and carbohydrates during autumn and winter as compared to controls.

Deficits were shown in spatial memory but not verbal memory. Regardless of month, C-SADs and S-SADs scored fewer correct items in the Visual Memory Scale than controls. C-SADs also scored fewer correct items than S-SADs. A possible explanation could relate to the disruption of circadian rhythms. The finding is indicative of abnormalities to the retino-hypothalamic pathway in both SAD groups that do not remit for summer. In addition, greater impairment was shown for C-SADs as compared to S-SADs that may relate to high levels of physiological arousal as reported on the TESI from additional stress, anxiety, and as symptoms of depression mood and ideation. Alternatively, the findings from spatial memory deficits show consistency with the dual vulnerability hypothesis that suggests that all SAD sufferers experience initial disturbances to sleep and energy levels followed by depression in those who are vulnerable. Deficits are evident for both SAD groups possibly relating to impairment in the visual pathway, with more extreme deficits in C-SADs indicative of their additional vulnerability to depression.

Only the C-SAD group showed right hemispheric deficits in the spatial version of Hemispheric Asymmetry with slower response times in April and June.

Further, regardless of group, participants all showed left hemisphere deficits in autumn/ winter for both verbal and spatial versions of Hemispheric Asymmetry.

Evidence from the trait version of the TESI shows the C-SAD group to feel greater tension stress from all four stressors (work, family, finance, and own body) at varying times of the year. However effort stress did not vary between the groups suggesting that C-SADs do not expend additional effort to cope with their greater stress load, thus producing greater negative emotion and increased depression symptoms. In contrast to the *trait* stress differences felt, the two SAD groups did not differ from each other in *current* felt stress

The findings from the TESI also show consistency with the dual vulnerability hypothesis. C-SADs showed greater overall stress from work and body during winter, as well as in December indicative of underlying characteristic traits of vulnerability to depression. Whilst all participants showed a seasonal pattern feeling less relaxed, and more anxious and sullen during April and June, overall C-SADs felt greater anxiety and anger than controls, and greater boredom than S-SADs. S-SADs felt more anger than controls. As in Longitudinal Study 1, C-SADs showed a combination of emotions suggesting hypo-arousal (e.g., boredom, sullenness) together with high physiological arousal (e.g., anxiety, anger).

Future directions

It is currently unclear whether the nature of memory deficits in SAD relate to a disruption of visual processing in the visual pathway or are a result of depressed mood ideation, for example, anxiety or an inability to concentrate. Two memory tasks from the Wechsler Memory Scale (Wechsler, 1987) were

administered for Longitudinal Study 2 of cognitive performance. Group differences were shown in the spatial memory task, Visual Memory Span but not in Digit Span, the verbal memory task, thus dissociating between verbal and spatial memory. Regardless of month, the SAD groups recalled significantly fewer correct items than the control group for both forward and backward conditions of Visual Memory Span. C-SADs also recalled significantly fewer correct items than S-SADs for both forward and backward conditions of Visual Memory Span. The finding suggests that disruption to visual processing may be present in the visual pathway of SAD sufferers. Further experimental research is needed to determine the abnormality in the visual pathway and may also implicate vulnerability to SAD. Differences between the two SAD groups suggest a vulnerability to the depressed mood ideation for C-SADs, but not for S-SADs. Further clarification of the nature of deficits to memory in the SAD groups may further determine a clear-cut distinction between clinical and subsyndromal levels of SAD.

Previous findings (Cohen et al., 1992; Volf et al., 1993) have suggested an asymmetrical hemispheric pattern specific to SAD, however while the present findings may be consistent with this suggestion, further research is required for confirmation of any hemispheric specialisation in SAD. Additional research with hemispheric specialisation tasks will further determine physiology in SAD and further determine the extent of vulnerability, thus allowing clearer distinction from NSD.

Conclusions

As in Longitudinal Study 1 of autonomic arousal, the current study has shown the specificity of depressed mood and associated atypical vegetative

behavioural symptoms to the autumn/ winter months, supporting the distinction of SAD from non-seasonal major depressive disorder.

Impairment to cognitive performance for the SAD groups may be distinct from any impairment that has been shown in NSD, though further confirmation is required. Deficits were shown to spatial memory in the SAD groups that are indicative of a disruption to visual processing in the visual pathway. More extreme deficits for C-SADs as compared to S-SADs may be the result of additional depressed mood and ideation characteristics such as anxiety. C-SADs showed right hemispheric deficits with slower response times in the spatial version of Hemispheric Asymmetry during April and June. All participants showed left hemispheric deficits in both verbal and spatial versions of Hemispheric Asymmetry in April and June.

Findings from the TESI show consistency with the dual vulnerability hypothesis. Seasonality was evident with all participants regardless of group reporting being less relaxed, and more anxious and sullen during autumn and winter giving support to the seasonal component of the dual vulnerability hypothesis. The C-SAD group show evidence of the depression component with greater felt tension though inadequate efforts to cope, and also lower positive emotions and greater negative emotions as compared to S-SADs during autumn and winter. More extreme emotions for the C-SAD group in December also suggest a vulnerability to depression. Trait characteristics evident for C-SADs that implicate vulnerability to depression is also distinct from 'current felt' stress which did not differ between the two SAD groups.

Seasonal variation was evident in several variables for control participants. Participants were more fatigued during winter. Left hemispheric impairment was shown with slower response times in autumn/ winter. Regardless of group, all participants exerted less effort to cope with tension stress from their own body during autumn/ winter. Positive emotions were rated lower, and negative emotions were rated higher in autumn and winter.

To summarise, this study adds further confirmation to seasonal changes in mood and associated atypical behavioural symptoms for the two SAD groups during autumn and winter. Impairment of cognitive performance has also been shown which may implicate vulnerability to SAD and also show distinction from NSD. Consistency has been demonstrated with the dual vulnerability hypothesis.

Part 111

Conclusions and future directions

Chapter 9

A summary of the research findings and future directions

Chapter 9: A summary of the research findings and future directions

Seasonality in the Tasmanian population

An initial component of the thesis, the epidemiological survey (Chapter 4) showed seasonal variation to mood and behavioural symptoms in a high percentage of the Tasmanian population. Up to nine percent may experience SAD at a clinical level, while a further twenty-six percent experience variation to mood and atypical vegetative symptoms at a subsyndromal level. Twenty four percent of the Tasmanian sample recorded little or no seasonality on the SPAQ. All remaining participants, comprising thirty-nine percent of the sample were categorised as mixed seasonals and included those who recorded variation to mood and associated behavioural symptoms that was not indicative of SAD. For example, the mixed seasonal group included those who experienced allergy difficulties at a particular time of year, those who experienced changes to diet and weight during seasonal sporting activity, and those who recorded changes to mood and atypical vegetative symptoms at a particular time of year such as Christmas or the university examination period.

Mood and behavioural symptoms

During the autumn and winter months all groups from the epidemiological survey showed seasonality to some degree with decreases to mood, social activity, and energy level, along with increases in eating, weight gain, and sleep. Spring and summer symptoms indicative of hypomania (e.g., increases in mood, social activity,

and energy level, and decreases in eating, weight gain, and sleep) were also evident in the two SAD groups. The SAD groups showed greater fluctuations between autumn/ winter and spring/ summer in symptoms than controls, with C-SADs showing more extreme fluctuations than S-SADs. Further support for hypomania in the Tasmanian sample was shown from hours of sleep in each season as recorded on the SPAQ. Compared to controls, the two SAD groups reported longer hours of sleep in autumn and winter consistent with the hypersomnia symptom for SAD, and less hours of sleep in summer suggesting hypomania (e.g., greater energy levels and social activity) for summer. In addition, the SAD groups reported feeling worst for the weather conditions 'short daylight', 'grey/ cloudy', and 'fog/ smog', and improving in mood with 'long daylight' and 'sunny days'.

Group differences were shown with C-SADs reporting a greater degree of seasonal change to all symptoms than S-SADs and controls. S-SADs also reported a greater degree of seasonal change than controls. A discriminant analysis classified high percentages of each group further confirming the distinction between the groups, firstly from the degree of seasonal change to symptoms, followed by the hours of sleep per season, then lastly the participants' perception of seasonal change/ disturbance as a problem in their lives.

Strong support was shown from the epidemiological survey for a metabolic involvement in seasonality to mood and associated atypical vegetative behavioural symptoms and/ or SAD. Several regression analyses and a factor analysis were conducted to gain an understanding of any relationships among the SPAQ variables. Whilst all symptoms were shown to be predictors in the global seasonality score

(GSS), the three symptoms, energy, weight, and sleep contributed 87% of the variance indicating a metabolic involvement in SAD. For global rating (GR), mood emerged as the best predictor followed by sleep, energy, and weight. The factor analysis conducted on the SPAQ variables gave a three-factor solution: a Seasonal Weather factor, a SAD factor, and a Metabolic factor. The SAD factor includes high loadings on mood, short daylight, GSS, and GR, while the metabolic factor has high loadings on variables involving sleep and weight. The factor structure also shows consistency with Young et al.'s (1991) dual vulnerability hypothesis that proposes that SAD sufferers experience seasonal change and/ or disturbances to the atypical vegetative symptoms, followed by depressive episodes in those who are vulnerable.

The aetiology in SAD is thought to be the shorter daylight hours during the autumn and winter months in locations of greater than latitude 40° N or S (Rosenthal & Wehr, 1992). A review of the literature involving seasonal variation shows SAD to be biologically based with disruptions to circadian rhythms (e.g., Hill, 1992), neurotransmitter abnormalities (e.g., Hill; Skwerer et al., 1989) as well as an ANS involvement (e.g., Putilov & Danilenko, 1998). Seasonal variation appeared to represent a continuum of severity in the general population ranging from little or no seasonality, through mild to moderate seasonal changes, with SAD being an exaggeration of seasonal disturbances to mood and associated atypical behavioural symptoms at the extreme end. SAD is currently categorised in the DSM IV (American Psychiatric Association, 1994) as a 'Seasonal Specifier' that may be applied to recurrent major depressive disorder. However accumulating evidence suggests that SAD may be a disorder quite distinct from non-seasonal depression. Hence the aim of this thesis was to determine the nature of SAD in relation to

seasonality in the general population. The epidemiological survey (Chapter 4) has confirmed group differentiation from the symptoms, implicated a metabolic involvement, and confirmed the 'short day' aetiology in SAD. However, due to the linear nature of the scale on the screening questionnaire SPAQ, confirmation of any continuum of severity in seasonal changes to mood and behavioural symptom was not possible.

The experimental component of the thesis included two longitudinal studies. Longitudinal Study 1 on autonomic arousal was conducted to seek evidence for hypo-arousal in SAD. The symptoms of SAD (e.g., hypersomnia, increases to eating, weight gain, and lack of energy) are consistent with an increase in parasympathetic tone. An investigatory study (Austen & Wilson, 2001), conducted as a forerunner to Longitudinal Study 1 showed an increase in vagal tone representing the sleep symptom supporting similarities between SAD and the hibernation process in animals. In contrast, studies (Balogh et al., 1993; Rechlin et al., 1994, 1995) have shown decreases in vagal tone in NSD. Hence it seems likely that underlying mechanisms in SAD differ from those in NSD. Psychophysiological measures from Longitudinal Study 1 also showed support for an increase in vagal tone with a decrease in HR. In addition, an increase in SCL represents hyper-arousal or an increase in sympathetic arousal. Longitudinal Study 2 of cognitive performance was conducted (a) to determine cognitive efficiency across the twelve-month period, and (b) to investigate whether any abnormality may be present in the visual pathway in SAD as well as investigating the extent of any right hemispheric involvement in SAD. Deficits for the SAD groups were evident in spatial memory but not verbal memory. The two SAD groups recalled significantly fewer correct

items than controls. The finding suggests abnormalities to the retino-hypothalamic pathway in SAD that do not remit for summer. In addition C-SADs showed a greater deficit in spatial memory than S-SADs that may be due to their high physiological arousal from additional stress, anxiety, and depressed mood. A group difference was evident in the spatial condition of the Hemispheric Asymmetry task with a right hemispheric impairment during winter for the C-SAD group, consistent with interference between processing spatial information and right hemisphere processing of negative emotions. Although there were no group differences, slower reaction times were also evident for winter in the Hemispheric Asymmetry and Mental Rotation tasks and will be described more fully in the section on documentation of circannual rhythms.

Emotion

Emotional tone was measured in both longitudinal studies using the Tension and Effort Stress Inventory (TESI). Even though testing sessions for Longitudinal Study 1 were conducted in January, March, May, July, September, and November, and testing sessions for Longitudinal Study 2 were conducted in February, April, June, August, October, and December, the results are consistent.

The two longitudinal studies showed the two SAD groups to feel greater tension stress as well as expending greater effort to cope with family, finance, and body, as compared to controls. Both longitudinal studies showed all groups to feel increased tension stress from work during the university semesters. However for effort stress, participants in Longitudinal Study 1 expended additional effort to cope, but did not for Longitudinal Study 2. It is most likely that the differences in

the months of testing account for this inconsistency. For Longitudinal Study 1, the testing months included March (early autumn prior to symptom onset), May, and July (mid-semester break). On the other hand, greater stress loads may have been expected during the testing months for Longitudinal Study 2 that included April (where seasonal symptoms onset was evident), June (university examination period), and August (commencement of workload in the university semester 2).

In both longitudinal studies, the C-SAD groups only felt greater tension stress for own body during autumn and winter months as compared to spring and summer, though any attempt to compensate through additional coping efforts was not evident. The additional stress is most likely related to the SAD symptoms of carbohydrate cravings and weight gain, with fatigue, lack of energy, and low self-esteem being factors in inadequate coping.

All participants showed winter increases in negative emotion and decreases in positive emotion regardless of group. Participants were less relaxed and excited, and showed greater levels of anxiety, sullenness, humiliation, and guilt during winter. Two low arousal emotions, placidity and sullenness were evident for autumn/ winter and are supportive of increased vagal tone implicating a state of torpor and with similarities to hibernation in some mammals. In contrast, increased arousal was also evident. For the SAD groups, there was also greater anxiety and anger than for controls, reflecting hyper-arousal or increased felt arousal in winter. The findings are consistent with the psychophysiological findings of increased vagal tone evidenced by a decrease in HR, together with increased sympathetic arousal evidenced by increased SCL.

Comparison between SAD and non-seasonal Major Depressive Disorder

Several findings offer support for the suggestion that SAD is a disorder distinct from NSD. The thesis sought to investigate the proposition that the symptoms of SAD represent an increased parasympathetic tone or hypo-arousal. Support for the proposition was shown from (a) a pilot study (Austen & Wilson, 2001) with increases in vagal tone in S-SAD participants during winter as compared to a non-winter period and comparison with control participants and (b) Longitudinal Study 1 where decreases in HR were shown in winter. The atypical vegetative symptoms of SAD (e.g., hypersomnia, increased eating and carbohydrate cravings, weight gain, lack of energy, and social withdrawal) differ from those typical in NSD (e.g., insomnia, decreases to eating, and weight loss) where decreased parasympathetic tone or hyper-arousal has been shown (e.g., Putilov & Danilenko, 1998; Rechlin et al., 1994).

In addition, the metabolic symptoms, energy, weight, and sleep, which together explain eighty seven percent of the variance in the GSS, also support the proposition of an increase in parasympathetic tone in SAD. Further, after mood, the three metabolic symptoms (energy, weight, and sleep) also contribute highly to the global rating (GR) or participants' overall perception of the seasonal disturbance in their lives. A Principal Components analysis produced a three-factor solution: a Seasonal Weather Factor, a SAD Factor, and a Metabolic Factor. The separation of mood in the SAD Factor, from the atypical vegetative symptoms in the Metabolic Factor added further support to SAD being a disorder distinct from NSD.

The shorter daylight during the autumn/ winter months is thought to be an aetiology that is specific to SAD. The weather condition 'short daylight' appeared as a consistent predictor for several SPAQ variables adding further support for its aetiology in SAD. These were: GSS (participants rating of six symptoms: mood, sleep, eating, weight, social activity, and energy), MSS (participants rating of the three metabolic symptoms: sleep, weight, and energy), and GR (participants perception of seasonal change as a problem in their lives).

Implications for theories in SAD

Early research in SAD suggested theories involving a specific pathophysiology, for example, the melatonin hypothesis, circadian theories, or theories involving the retino-hypothalamic pathway. A summary of these theories is outlined in Chapter 6. More recent literature suggests that more than one mechanism may be involved in the pathophysiology of SAD. For example, Putilov and Danilenko (1998) argue that a combination of effects including circadian rhythm, sleep, and energy regulation, and the sympatho-adrenal systems all have an involvement in SAD. Several findings from this thesis show consistency with findings of Putilov and Danilenko. Firstly, the metabolic symptoms (sleep, energy, and weight) were shown to contribute eighty seven percent of the variance in SPAQ SAD symptoms. Secondly, increases to vagal tone were shown from a pilot study (Austen & Wilson, 2001) as well as in Longitudinal Study 1 on autonomic arousal. These two findings suggest hypo-arousal or a state of torpor showing similarities with the hibernation process in some animals.

The thesis findings are also consistent with the dual vulnerability hypothesis that suggests initial seasonal disturbances to sleep and energy, followed by depression in vulnerable individuals (Young et al., 1991). The results from the TESI showing the experience of arousal indicate that both high and low arousal emotion have an involvement in SAD. Two low arousal emotions, placidity and sullenness were shown to increase in winter as compared to summer and this is consistent with hypersomnia and a lack of energy thus giving a seasonal component. Placidity may in turn lead to feelings of anxiety and anger due to a build up of uncompleted work, social commitments, and increased arousal. Guilt, a transactional high arousal emotion may represent not fulfilling expectations of others, or letting others down. Anxiety, anger, and guilt were increased in winter and are indicative of a depression component in SAD. Of the emotions experienced during winter, placidity is the only pleasant emotion, indicating a tendency to remain calm and not worry about things. It is possible that the seasonal component of SAD is an extreme of the seasonal changes to mood and associated atypical vegetative symptoms evident in the general population. Further, those who are vulnerable, experience depression in addition to their seasonal disturbances, and suffer SAD at a clinical level.

Documentation of circannual rhythms

From the two longitudinal studies seasonal variation was documented for several variables, thus adding to current knowledge on circannual rhythms. For some variables, circannual rhythmicity was reported for the first time, while for other responses circannual rhythmicity was shown to support previous findings.

Many SAD-like symptoms showed seasonal variation in control participants, demonstrating mood and associated atypical vegetative behavioural symptoms vary with the seasons in the general population. For Longitudinal Study 1, all participants showed an increased depressed mood and ideation and decreased hypomania mood and ideation in May as indexed by the WMI. For both longitudinal studies, participants were more fatigued during autumn and winter as compared to spring and summer. All participants in Longitudinal Study 2 showed a preference for carbohydrates during autumn/ winter. Body mass index was higher for all participants in July as compared to March and September most likely reflecting the additional carbohydrate consumption. Thus the tendency for decreases to mood along with associated atypical behavioural symptoms in winter (e.g., Kasper, 1989; Terman, 1989) has also been shown in the current research with participants from Tasmania, Australia's southern most state.

Several physiological measures were also shown to vary with the seasons in control participants. Predicted RSA scores, not previously measured across a twelve-month cycle, were higher for March, May, July, and September as compared to January, thus documenting seasonality for the first time. Even though no change was evident to actual RSA, the dynamical properties of the RSA system appear to have changed. It seems that RSA output was maintained through accommodation processes in the vagal system despite changes in functional dynamics. HR was lower in autumn as compared to November, further supporting the pilot study (Austen & Wilson, 2001). Lacoste and Wirz-Justice (1989) showed no seasonal variation in baseline HR. Controls showed an increased SCL also supporting the

hyper-arousal noted by Austen and Wilson as well as previous findings (Neumann, 1968; Venables & Christie, 1973). Lower skin temperatures were recorded during winter than in spring/ summer also consistent with previous findings (Gardner-Medwin et al., 2001).

Regardless of group, impairment was shown for winter in the reaction time tasks. A slower reaction time was evident for the left hemisphere in the verbal condition of Hemispheric Asymmetry in autumn and winter as compared to spring and summer, indicating that with verbal processing in the left hemisphere was associated with slower responding during winter. Similarly, a slower reaction time during winter was evident for all participants for the Mental Rotation task, reflecting a slowing of spatial processing in the right hemisphere during winter.

Several emotions from the TESI varied with the seasons in control participants in both longitudinal studies. Control participants felt less relaxed but more sullen during autumn and winter. Two transactional emotions, shame and guilt, were also increased for controls during autumn and winter. Controls also varied with the seasons in motivational tendencies according to scales from the MSP, with higher scores on the emotionality/ effortfulness scale in summer compared to winter. Controls were also more optimistic in summer than in winter, though they did not vary in pessimism. Both findings are consistent with a state of hypo-arousal and the tendency for hypersomnia, fatigue, and lowered energy levels evident in the general population during winter. The seasonal variation to motivational styles and tendencies also suggest that controls adjust over time in order to cope with their circumstances (Apter & Desselles, 2001).

The documentation of circannual rhythms has implications for research methodology in controlling for any experimentation using repeated measures across time and/ or seasons, as well as for the accuracy and meaningfulness of interpretations of comparison of between group measures taken at different times of the year. In addition, SAD-like seasonal symptoms in the general population during winter, for example, hypersomnia, fatigue, lack of energy, and weight gain, together with the tendency for negative emotion, has implications for efficiency and adjustment in the workplace, and in any situation involving performance evaluation, testing procedures, and examination performance during winter.

Limitations

Some limitations of the study require mention. A methodological concern in conducting any repeated measures study across time, either involving time of day or season, requires additional control measures. Due to circadian differences in physiological response levels as well as performance measures, a difficulty remained in controlling for time of day during testing. Counterbalancing in the longitudinal studies included testing half of the participants in the morning (8.00 a.m. to 12 noon) and half in the afternoon (2.00 p.m. to 6.00 p.m.). Participants were tested on six occasions at bimonthly intervals, with testing time being approximately the same time at each testing session. During this twelve-month period of testing, many individuals experienced problems that included personal problems, relation difficulty, work stress, financial and family pressures.

In addition, some variables showed differences between the two longitudinal studies. The two studies were conducted concurrently, with laboratory testing for Longitudinal Study 1 of autonomic arousal being conducted in January, March, May, July, September, and November. Laboratory testing for Longitudinal Study 2 of cognitive performance was conducted in February, April, June, August, October, and December. Differences to group symptom pattern are evident between the two longitudinal studies and may simply reflect the differing months of laboratory testing. For example, testing may reflect the additional stress of examination in November and June. Also, participants may show a tendency for relaxation, and lower stress levels during the holiday periods in December/ January (summer) as well as in the university vacation in July (winter). On the other hand, studies conducted in the northern hemisphere, with a mid-winter Christmas in December are confounded by additional eating and thus weight gain during the festive holiday season.

Regardless of these limitations, the current thesis is the first study to have taken extensive measures including vagal tone in SAD and control participants across a twelve-month cycle.

Future directions

Several areas for future research have been revealed from findings evident from the thesis. Firstly, the thesis has shown a high metabolic involvement in seasonal variation to mood and associated atypical vegetative symptoms, along with increases to vagal tone, which support similarities with the hibernation process in

animals. Further examination of autonomic measures in SAD, in particular, energy, sleep, and vagal tone, along with biochemical measures including catecholamine, noradrenaline, adrenaline, and blood lipids should offer further confirmation of the presence of hypo-arousal or torpor-like state thus enabling further comparison between SAD and hibernation.

Secondly, further experimentation of cognitive performance will clarify any abnormality in the retino-hypothalamic pathway with implications for understanding why some individuals are vulnerable to suffering SAD. It is currently unclear whether spatial deficits to cognitive performance in SAD participants relate to disruption in the retino-hypothalamic pathway, or are as a result of depressed mood, and/ or increased anxiety and stress levels in winter. Additional experimentation with spatial tasks will also assist in understanding the nature of any deficits in the SAD groups as well as any hemispheric asymmetry specific to SAD. Also, clarification of right hemispheric function in SAD may further assist in a distinction between SAD and NSD.

For further confirmation of 'dual vulnerability' in SAD, a clear-cut distinction between subsyndromal and clinical levels is required. Further clarification may also show any underlying abnormality in the visual pathway of SAD participants and further assist in understanding the mechanisms underlying SAD.

An additional area relating to seasonality in the general population has emerged from the thesis. Several findings from the study implicate decreases to performance during winter (e.g., increased sleeping, fatigue, and slowed reaction time in cognitive tasks), as well as other times of additional stress loads. For example, increases to HR, SCL, and anxiety during November at the time of end of the university examination period. While the additional stress load can be seen as a limitation of the study, on the other hand, the findings suggest an area for future research in determining physiological and emotional arousal levels during evaluation or examination periods, as well as implications for achievement levels. The finding also has implications for performance and/ or production output in the workplace during winter.

Conclusions

The thesis has shown decreases to mood and associated atypical vegetative symptoms (hypersomnia, fatigue, overeating, weight gain, and loss of energy) in the two SAD groups that were specific to the autumn and winter seasons, as well as the remission of symptoms in spring and summer. In addition, the atypical vegetative symptoms were found in control participants to a certain degree.

A comparison was made between SAD and the hibernation process in animals (Chapter 6) that warranted further investigation. The thesis has shown the importance of the metabolic symptoms (energy, sleep, and weight) in SAD, thus showing consistency with a torpor-like state or hibernation and differentiating SAD from non-seasonal depression. In addition, increased vagal parasympathetic tone

shown in a pilot study (Austen & Wilson, 2001) was supported by decreased HR during winter in the longitudinal study on autonomic arousal. The increases to vagal tone, together with increases in the emotions of placidity and sullenness during winter represent a state of hypo-arousal. In contrast to this hypo-arousal, SCL showed increases during winter, and there were increases in the emotions of anger and anxiety, representing hyper-arousal or increased physiological arousal. The parasympathetic activation appears contradictory to the increased sympathetic tone, but could indicate cyclic changes. Low arousal may be associated with reduced physical activity and a failure to meet goals that produces stress and high arousal, leading back to low arousal from reduced motivation and fatigue.

The results are consistent with dual mechanisms underlying SAD. Firstly, a seasonal component that may be on a continuum from those who experience little or no seasonality through to those who experience extreme seasonal disturbances to the atypical vegetative symptoms. Secondly, a depression component is evident in those who are vulnerable, creating more extreme disturbances to mood and atypical vegetative symptoms during winter.

Evidence was sought from the study regarding the continuum of seasonality. Several findings suggest that this may not be the case, but rather separate factors for seasonality and depression strongly supporting the operation of a dual vulnerability mechanism. Any stronger support for such theory requires being able to make a clear-cut distinction between subsyndromal and clinical levels in SAD.

Impairment was shown to cognitive performance in Longitudinal Study 2. The two SAD groups showed deficits to spatial memory that did not vary with the months, showing consistency with impairment in the visual pathway that did not remit for summer. The finding was not evident for verbal memory. The deficit was greater for C-SADs than for S-SADs that may relate to the additional physiological arousal from greater tension stress and anxiety. All participants showed left hemisphere deficits in both verbal and spatial versions of the Hemispheric Assymetry task during autumn and winter. In addition the C-SAD group showed right hemisphere deficits in the spatial version of Hemispheric Assymetry during autumn and winter.

Several variables were shown to vary with the seasons in the control group thus extending current knowledge with the documentation of circannual rhythms. The tendency for decreases in mood along with associated atypical behavioural symptoms was evident for autumn/ winter. Fatigue, a preference for carbohydrates, and BMI were also increased for winter. Several physiological measures varied with the seasons. Predicted RSA and SCL were increased, while HR and skin temperature were decreased in winter. Deficits were shown to cognitive performance with slower response times evident in the Hemispheric Asymmetry and Mental Rotation tasks in winter. A tendency was also shown for decreased positive emotion and increased negative emotion in winter. The general population show physiological, psychological, and performance differences with time of year.

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Appendix A

Seasonal Pattern Assessment Questionnaires:

Version 1: Screening questionnaire used in epidemiological survey

Version 2: Screening questionnaire used in participant selection

SEASONAL PATTERN ASSESSMENT QUESTIONNAIRE

NAME.....AGE.....M/F.....
NUMBER OF YEARS RESIDENCE IN TASMANIA.....

We are interested in the degree to which people's feelings/behaviour may or may not change with the four seasons that we experience during a year. This questionnaire describes the degree of change that may be experienced with seasonal weather conditions and then asks you to rate these behavioural/feeling changes according to a rating scale.

A.. Below is a scale that indicates the degree of change that may accompany feelings and behaviours with each season. Indicate the degree to which you consider that the following feelings/behaviours may be affected by seasonal changes (e.g., spring to summer, summer to winter, etc.). Place a cross (X) through the appropriate place on the line scale below.

- 1 = no change
2 = slight change
3 = moderate change
4 = marked change
5 = extremely marked change

Sleep Length. 1 2 3 4 5
Social Activity 1 2 3 4 5
Mood 1 2 3 4 5
Weight 1 2 3 4 5
Appetite 1 2 3 4 5
Energy Level. 1 2 3 4 5

B. Below is a list of feelings and behaviours that may change with each season. Please read each item carefully and indicate the months of the year in which you experience these feelings/behaviours by placing a cross (X) in the appropriate space that corresponds to the month (s). If the feeling/behaviour is not applicable to you, place a cross (X) in the N/A column.

JAN FEB MARCH APRIL MAY JUNE JULY AUG SEPT OCT NOV DEC N/A

feel best.....
feel worst.....
lose most weight.....
gain most weight.....
sleep most.....
sleep least.....
eat most.....
eat least.....
socialize most.....
socialize least.....

C. Below is a scale that indicates the degree to which your mood/energy levels may change with different weather conditions. Please read each item carefully and indicate the degree to which each of the different weather conditions affect your mood/energy level by placing a cross (X) through the appropriate place on the line scale below.

- 1 = makes you feel in very low spirits or markedly slowed down
2 = makes you feel in moderately low spirits or moderately slowed down
3 = makes you feel in slightly low spirits or slightly slowed down
4 = no effect
5 = slightly improves your mood or energy level
6 = moderately improves your mood or energy level
7 = markedly improves your mood or energy level

Cold	1	2	3	4	5	6	7
Hot	1	2	3	4	5	6	7
Humid	1	2	3	4	5	6	7
Sunny	1	2	3	4	5	6	7
Dry	1	2	3	4	5	6	7
Grey and Cloudy	1	2	3	4	5	6	7
Long daylight	1	2	3	4	5	6	7
High Pollen Count	1	2	3	4	5	6	7
Foggy and Smoggy	1	2	3	4	5	6	7
Short daylight	1	2	3	4	5	6	7

D. Indicate, by placing a cross (X) in the appropriate box, the degree to which your weight fluctuates during the year.

0 - 2 kg ☐ 4 - 5 kg ☐ 8 - 10kg ☐
2 - 3 kg ☐ 6 - 7 kg ☐ over 10kg ☐

E. Estimate to the nearest half hour, the number of hours sleep that you require in each 24hour period during each of the four seasons represented below :

Autumn (March 20 - June 20).....
Winter (June 21 - Sept 20).....
Spring (Sept 21 - Dec 20).....
Summer (Dec 21 - March 20).....

F.. If you experience changes with the seasons do you feel that the problem is :
(place a cross (X) in the box).

mild ☐ marked ☐ disabling ☐
moderate ☐ severe ☐

contact phone number.....

Contact phone number

Appendix B

**Written signed consent forms signed by participants in
Longitudinal Study 1 - Autonomic Arousal in SAD and
Longitudinal Study 2 - Cognitive Performance**



Seasonal variation in physiological arousal and mood in seasonal affective disorder and controls

The above investigation will be undertaken by Mrs. Margaret Austen under the supervision of Dr. George Wilson in the Department of Psychology. The research aims to determine the extent of seasonal changes across a twelve month period on physiological and psychological indices in seasonal disturbance at subsyndromal and clinical levels. The study may also reveal the physiological processes involved in seasonal disturbance with implications for control and treatment of the condition. The study will involve six sessions in the Psychophysiology Laboratory at bimonthly intervals (March, May, July, September, November, and January). Each session will take approximately 45 - 60 minutes. Physiological measures will be height, weight, skinfold thickness, sublingual and skin temperature, heart rate, respiration, and skin conductance. Psychological indices will determine any extent of mood variation. Standard laboratory procedures will be used. These do not present any risk to participants.

Participation in the research study is voluntary. A participant may withdraw at any time without prejudice, simply by stating the desire not to continue. The aggregated results of the whole study only will be used for research purposes and may be reported in scientific journals. All data will be kept in a confidential manner. Individual results will not be released to any person, except at that person's request. The research has received ethical approval from the University Ethics Committee (Human Experimentation) and complies with the laws of the state. Any questions relating to the research may be answered by contacting Margaret Austen, ph 6220 2246, or Dr George Wilson, ph 6220 2240. Concerns of an ethical nature or complaints about the manner in which the project is conducted may be directed to the Chair (Dr Margaret Otlowski, ph 6220 7569) or Executive Officer (Chris Hooper, ph 6220 2763) of the University Ethics Committee (Human Experimentation). Ethical or personal concerns related to the study may be discussed confidentially with a University Student Counsellor, ph 6220 2099.

"I have read the information above and any questions I have asked have been answered to my satisfaction. I agree to participate in this investigation and understand that I may withdraw at any time without prejudice. I agree that research data gathered for the study may be published provided that I cannot be identified as a subject."

Name of subject

Signature of subject Date

"I have explained this project and the implications of participation in it to this volunteer and I believe that the consent is informed and that he/she understands the implications of participation."

Signature of investigator Date



Seasonal variation in cognitive efficiency and mood in seasonal affective disorder and controls

The above investigation will be undertaken by Mrs Margaret Austen under the supervision of Dr George Wilson in the Department of Psychology. The research aims to determine the extent of seasonal changes to cognitive performance in seasonal disturbance at subsyndromal and clinical levels. The study will involve six sessions in the Psychophysiological Laboratory at bimonthly intervals (April, June, August, October, December, and February). Each session will take approximately 45-60 minutes. Cognitive performance will examine learning, memory, and visuo-spatial efficiency. Psychological indices will determine any extent of mood variation. Standard laboratory procedures will be used. These do not present any risk to participants.

Participation in the research study is voluntary. A participant may withdraw at any time without prejudice, simply by stating the desire not to continue. The aggregated results of the whole study only will be used for research purposes and may be reported in scientific journals. All data will be kept in a confidential manner. Individual results will not be released to any person, except at that person's request. The research has received ethical approval from the University Ethics Committee (Human Experimentation) and complies with the laws of the state. Concerns of an ethical nature or complaints about the manner in which the project is conducted may be directed to the Chair (Dr Margaret Otlowski, Ph 6220 7569) or Executive Officer (Ms. Chris Hooper, Ph 6220 2763) of the University Ethics Committee (Human Experimentation). Personal concerns related to the study may be discussed confidentially with a University Student Counsellor, Ph 6220 2099, if required. A copy of this information is available for each participant. Any questions relating to the research may be answered by contacting Mrs Margaret Austen, Ph 6220 2246, or Dr George Wilson, Ph 6220 2240.

"I have read the information above and any questions I have asked have been answered to my satisfaction. I agree to participate in this investigation and understand that I may withdraw at any time without prejudice. I agree that research data gathered for the study may be published provided that I cannot be identified as a subject."

Name of subject

Signature of subject Date

"I have explained this project and the implications of participation in it to this volunteer and I believe that the consent is informed and that he/she understands the implications of participation."

Signature of investigator Date

Appendix C

Health checklist completed by all participants

HEALTH CHECKLIST

Name:

Age:

Gender (please circle) M / F

1. Have you at present, or have you in the past, had any cardiac problems? (For example, palpitations, or high blood pressure) YES / NO

If YES, please give more detail
.....
.....

2. Have you at present, or have you in the past, had any respiratory problems? (For example, asthma, or bronchitis) YES / NO

If YES, please give more detail
.....
.....

3. Are you currently on any medication? YES / NO

If YES, please give more detail
.....
.....

4. Do you smoke? YES / NO

If YES, how many packets on average would you smoke per week?

5. How much caffeine would you partake in an average day? Please indicate the type and amount
.....

6. What is your resting heart rate (if known)?

7. Are there any other details which may be relevant to your participation in this study?
.....
.....

Appendix D

Addendum to the Beck Depression Inventory

used to assess atypical vegetative symptoms of depression

Addendum of the Beck Depression Inventory

22

- 0 I can sleep as well as usual
- 1 I sleep longer than usual
- 2 I wake up 1-2 hours later than usual
- 3 I wake up more than 2 hours later than usual

23

- 0 My appetite is not worse than usual
- 1 I have more appetite than usual
- 2 My appetite is much greater than usual
- 3 I can eat all day

24

- 0 My weight has not increased lately
- 1 I have gained more than 2 kilos
- 2 I have gained more than 5 kilos
- 3 I have gained more than 15 kilos

Appendix E

**Weekly Mood Inventory: a questionnaire used to
determine any extent of seasonal atypical behavioural symptoms**

DIRECTIONS Please note that all information will be treated as confidential
This form is designed to find out how you have been feeling and functioning *during the past week*. Indicate, by placing a cross (X) in the appropriate box, how you have been feeling/functioning. After answering the questions on this page, please answer the scaled questions on page 2.

DURING THE PAST WEEK :

1. Have you taken any medications ?

- ☐ No
☐ Yes, no change since previous week
☐ Yes, changed since previous week ⇒

Medications & Dosage :

2. Have you had a cold, flu, or any illness ?

- ☐ No
☐ Yes ⇒

Please Specify :

3. Has anything happened during the past week which might have affected your feelings or functioning ?

- ☐ No
☐ Yes ⇒

Please Specify :

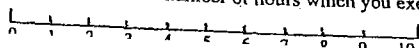
4. For menstruating females only : Have you menstruated this week ?

- ☐ No
☐ Yes

When did you last menstruate ?

Date began.....
Date ended.....

Please indicate on the scale below the number of hours which you exercise in an average week.



Please read the following items carefully and place a cross (X) in the box that best describes how you have been feeling *in the past week*.

1. I have experienced sad feelings
2. I have felt hopeful about the future
3. I have felt like crying
4. I have experienced sleep interruptions
5. I have felt guilty
6. I have felt happy or elated.
7. I have been sleeping less than usual
8. I have been waking in the early hours of the morning
9. I have been efficient at work
10. I have had a great need for sleep
11. I have felt energetic
12. I have felt that life is not worth living
13. I have had a desire for sweets and starches
14. I have wanted to socialize with others
15. I have been gaining weight
16. I feel I have been looking good
17. I have had difficulty getting to sleep
18. I have felt creative
19. I have felt disappointed in myself
20. I have had a large appetite
21. I have had an interest in sex
22. I have been able to think quickly
23. I have been losing weight
24. I have been sleeping more than usual
25. I have had a hard time making decisions
26. I have been able to enjoy things

	not at all							very much	
1	2	3	4	5	6	7			
2	3	4	5	6	7				
3	2	3	4	5	6	7			
4	2	3	4	5	6	7			
5	2	3	4	5	6	7			
6	2	3	4	5	6	7			
7	2	3	4	5	6	7			
8	2	3	4	5	6	7			
9	2	3	4	5	6	7			
10	2	3	4	5	6	7			
11	2	3	4	5	6	7			
12	2	3	4	5	6	7			
13	2	3	4	5	6	7			
14	2	3	4	5	6	7			
15	2	3	4	5	6	7			
16	2	3	4	5	6	7			
17	2	3	4	5	6	7			
18	2	3	4	5	6	7			
19	2	3	4	5	6	7			
20	2	3	4	5	6	7			
21	2	3	4	5	6	7			
22	2	3	4	5	6	7			
23	2	3	4	5	6	7			
24	2	3	4	5	6	7			
25	2	3	4	5	6	7			
26	2	3	4	5	6	7			

Appendix F

Sleep Quality and Food Preference Questionnaire:

A questionnaire to determine any changes

to sleep pattern and food preference

Sleep Quality and Food Preference Questionnaire

For each of the questions below indicate by circling the appropriate figure to represent how you have felt over the past week.

1. Have you experienced good nights of sleep over the past week?

Good nights

Bad nights

1 2 3 4 5 6 7

2. Have you been sleeping restfully over the past week?

Restful sleep

Disturbed sleep

1 2 3 4 5 6 7

3. Have you slept deeply over the past week?

Slept lightly

Slept deeply

1	2	3	4	5	6	7
---	---	---	---	---	---	---

4. Have you felt refreshed from sleep over the past week?

Feel refreshed

Feel fatigued

1	2	3	4	5	6	7
---	---	---	---	---	---	---

5. Have you noticed a change in food preference during the past week? **YES / NO**

If yes, do you consider this change to be: (CIRCLE ONE OR MORE)

- Fats
- Protein
- Carbohydrates
- Other

Appendix G

Journal publication of preliminary study conducted as a forerunner to

Longitudinal Study 1 - Autonomic Arousal in SAD

Austen, M. L. & Wilson, G. V. (2001). Increased vagal tone in subsyndromal SAD during winter. *Biological Psychiatry*, **50**, 28-34.

Increased Vagal Tone during Winter in Subsyndromal Seasonal Affective Disorder

Margaret L. Austen and George V. Wilson

Background: Seasonal affective disorder (SAD) is characterized by recurrent winter depression with summer remissions and/or hypomania. Further symptoms include hypersomnia, increased appetite, weight gain, fatigue, and social withdrawal, which may indicate autonomic changes during winter.

Methods: Measurements of respiratory sinus arrhythmia, heart rate (HR), and skin conductance level (SCL) were taken from 32 participants in subsyndromal SAD and control groups (eight male and eight female subjects in each group) in autumn and winter to determine any change in autonomic function. Measures were taken at baseline and during two stressor tasks. Single determinations of blood pressure, sublingual temperature, depression, aerobic fitness, and body mass index were also measured at each session. Replication in a second data collection period over subsequent winter and spring periods was conducted with an additional 32 participants to extend the findings and to counterbalance order effects in testing. Data were combined to produce "winter" and "nonwinter" test periods and statistically corrected for testing order.

Results: Respiratory sinus arrhythmia differences indicated that SAD subjects have increased vagal tone in winter. Both groups show a decrease for HR and increases for SCL and diastolic blood pressure in winter.

Conclusions: Seasonal affective disorder may show similarities with hibernation, and the results may indicate mechanisms different from those of nonseasonal depression. *Biol Psychiatry* 2001;50:28-34 © 2001 Society of Biological Psychiatry

Key Words: Seasonal affective disorder, seasonality, autonomic nervous system, respiratory sinus arrhythmia, heart rate, vagal tone

Introduction

Seasonal affective disorder (SAD) was first described as a clinical entity by Rosenthal et al (1984), although seasonal changes in affective disorder have been recorded since Hippocrates' time in 400 BC (Wehr 1989). Seasonal affective disorder is characterized by recurrent autumn-winter depressions with spring and summer remissions and/or hypomania. Further symptoms include hypersomnia, fatigue, increased appetite with carbohydrate craving, weight gain, and social withdrawal (Rosenthal et al 1984). Seasonal affective disorder may also be experienced at a subsyndromal level with depressive episodes to a lesser degree. Rosenthal and colleagues' (Rosenthal et al 1984) criteria for SAD include that the depression should not relate to any obvious effect of season-related psychosocial stressors.

Although the etiology of SAD is unclear, the reduced daily photoperiod in the autumn and winter months triggers this disorder in susceptible individuals. The incidence is higher at latitudes of greater than 40° (Rosenthal et al 1984). Neurotransmitters are involved in SAD, with noradrenaline, melatonin, and serotonin levels being reduced in SAD patients relative to normal subjects (Hill 1992; Shafi and Shafi 1990; Skwerer et al 1989). Several physiologic and psychologic variables that are involved in SAD have also been reported to show seasonal variation in the normal population (Lacoste and Wirz-Justice 1989). For example, blood pressure peaks in winter, though resting heart rate (HR) does not vary (Lacoste and Wirz-Justice 1989). Increases in metabolism (Lacoste and Wirz-Justice 1989) and skin conductance level (SCL) (Neumann 1968) have also been shown in winter.

The atypical vegetative symptoms of SAD differ from those typical of nonseasonal depression (e.g., insomnia, decreased appetite, weight loss) (American Psychiatric Association 1994). The symptoms of SAD (e.g., hypersomnia, increased appetite, weight gain) appear consistent with the effect of increased parasympathetic or decreased sympathetic tone. Thus it is possible that SAD patients have an inappropriate autonomic nervous system response to winter onset. Respiratory sinus arrhythmia (RSA) is recognized as a pure measure of parasympathetic nervous

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system activity that is sensitive to behavioral and cognitive changes (Bernston et al 1993; Porges and Byrne 1992). For example, RSA is enhanced with aerobic fitness (de Geus et al 1990), high during sleep, and reduced under conditions of stressful task performance (Porges 1995). On the other hand, HR (e.g., Porges and Byrne 1992), innervated by both the sympathetic and parasympathetic nervous systems, and SCL (e.g., Boucsein 1992), innervated solely by the sympathetic nervous system, typically increase under stressful task performance.

It remains unclear as to why some individuals show an increased vulnerability to the effects of changing seasons. Hobart, Tasmania, with a latitude of 43° south, is ideally situated to conduct studies with the purpose of investigating mood and physiologic arousal across two seasons (autumn and winter, or winter and spring) in subsyndromal SAD participants and in control subjects who show no seasonal disturbance.

The measurement of RSA, HR, and SCL in SAD and control participants in both winter and the other seasons should indicate any differences in autonomic activity. Additional constitutional and autonomic response measures were taken to support the proposition as well as to confirm previous findings. These were body mass index (BMI), systolic and diastolic blood pressure, and sublingual temperature. Aerobic fitness was measured using a bicycle ergometer test to control for any differential effect of fitness on RSA and HR between groups (de Geus et al 1990).

Mood and seasonal symptom ratings were predicted to be greater in SAD for winter and to indicate remission from symptoms during nonwinter periods. Respiratory sinus arrhythmia was predicted to be higher in winter and HR lower in winter for SAD subjects, indicating greater vagal tone. Respiratory sinus arrhythmia was predicted to decrease, and HR and SCL to increase, with cognitive stressor conditions for all participants irrespective of season or group. A higher BMI in SAD for winter was predicted, reflecting the weight gain symptom. A lower blood pressure may be expected for SAD subjects in winter, indicative of increased parasympathetic tone. A winter maximum in skin conductance, blood pressure, and temperature was expected for control subjects to confirm previous findings.

Methods and Materials

Participants

PARTICIPANT SELECTION. Psychology 1 students completed the Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal et al 1984), which screens seasonal change in mood and behavior. The sum of six (sleep length, social activity, mood, weight, appetite, and energy level) five-point scales ranging from

0 (no change) to 4 (extremely marked change) gave a global seasonality score (GSS). Subsyndromal SAD participants were selected on the basis of a moderate to high GSS together with their reported peak of symptoms in the winter months. Participants also satisfied Rosenthal et al (1984) criteria for the SAD symptoms with depressions that did not meet criteria for a major depressive episode (DSM-IV; American Psychiatric Association 1994). Control participants were selected on the basis of a low GSS and not responding with changes in mood and behavior across the seasons.

There were 64 participants, 32 (eight male and eight female in each group) who completed the autumn/winter data collection period and 32 (eight male and eight female in each group) who completed the second winter/spring data collection period. Course credit was gained for participation. All participants were Tasmanian residents for the past 3 years, to control for climatic adjustment at 43° south latitude. Ethical approval was obtained from the university's Human Research Ethics Committee and written informed consent was obtained from participants.

AUTUMN AND WINTER DATA COLLECTION PERIOD. Mean GSSs were 13.94 (SD 2.78, range 9–17) for subsyndromal SAD participants and 4.34 (SD 1.30, range 2–6) for control subjects. Mean ages for SAD and control groups were 19.3 and 20.4 years, respectively. Two control subjects (one male and one female) who recorded no seasonal complaint on the SPAQ were excluded from statistical analyses after scoring high seasonal depression during a laboratory testing session.

WINTER AND SPRING DATA COLLECTION PERIOD. Mean GSSs were 11.88 (SD 2.18, range 8–15) for subsyndromal SAD participants and 3.34 (SD 1.92, range 0–6) for control groups. Mean ages for SAD and control groups were 20.5 and 19.8 years, respectively. Winter results were excluded from statistical analyses for five participants (two control and three SAD) who did not complete the spring testing.

Materials

BECK DEPRESSION INVENTORY WITH ADDENDUM (BDI-ADD). The Beck Depression Inventory (Beck and Steer 1987) is a self-rating scale for depression. An addendum for assessing seasonal depression (Meesters and Jansen 1993; Y. Meesters and J.H.C. Jansen, personal communication, March 29, 1995) included items 12 (social withdrawal) and 17 (fatigability) from the BDI as well as items 16 (hypersomnia), 18 (appetite), and 19 (weight gain), which were inversely formulated.

WEEKLY MOOD INVENTORY (WMI). The Weekly Mood Inventory (Rosenthal et al 1989) is a self-rated seven-point scale developed for the assessment of SAD patients and requesting information about major life events, illness, and mood during the previous week. Factors of Depressed Mood and Ideation (DMI), Atypical Vegetative Symptoms (AVS), Typical Vegetative Symptoms (TVS), and Hypomania Mood and Ideation (HMI) were based on the clinical findings of others. The internal cohesiveness of the factors is high, ranging from .76 to .89 (Rosenthal et al 1989).

PSYCHOPHYSIOLOGICAL MEASURES. Psychophysiology recordings used a MacLab Data Acquisition System configured to record electrocardiograms (ECGs), cardiometer, respiration, and SCL. Electrocardiogram standard Ag/AgCl electrodes were filled with electrode gel and attached to the L-rib, R-rib placements and mastoid earth. A BioAmplifier coupler (AOInstruments, Sydney, Australia) with a bandpass filter of 0.3–50 Hz was used for ECG. Beat-to-beat cardiometer readings were computed directly from the ECG channel. Ag/AgCl electrodes for skin conductance were filled with electrode gel and attached to the first phalanx of first and third fingers on the subject's nondominant hand. A 0.5-V constant voltage bridge circuit was used to measure SCL, with output of the bridge applied to the MacLab (Lykken and Venables, 1971) with a bandpass filter of DC to 10 Hz. A Pneumotrace (AOInstruments, Sydney, Australia) Respiratory Chest Transducer was fitted around the participant's upper chest during full inspiration and then connected to a general purpose amplifier for respiration recording with a bandpass filter of DC to 50 Hz. Sampling rates were 200/sec for the ECG and cardiometer channels, 10/sec for SCL, and 100/sec for respiration. Respiratory pacing (see Procedure) used a four-channel interval generator delivering two tones for inspiration and expiration (1 kHz and 1.5 kHz) using Sonalerts. Each complete cycle was 6 sec (10 breaths/min).

CONSTITUTIONAL MEASURES. Height (m) and weight (kg) were measured using a KAWE (Salzkotten, Germany) wall-mounted height measure and Tanita (Tokyo) electronic digital scales, respectively. Blood pressure was measured using a Seim (Koyang, South Korea) portable Digital Blood Pressure Meter (Model SE-2000) and sublingual temperature taken using a Sharp (Osaka, Japan) Digital thermometer (Model MT 20). Submaximal fitness level was assessed by a 6-min period of exercise on a Repco (Melbourne, Australia) bicycle ergometer.

STRESSOR TASKS. The psychophysiological stressors in the experiment were the Digit Span subtest from the Wechsler Adult Intelligence Scale Revised (WAIS-R; Wechsler 1981) and Stroop Color Word Test (SCWT) (Stroop 1935). After practice with 40 stimuli, 100 stimuli were given in four blocks. Of these, 40 were the same (e.g., the word *red* and red ink) and 60 were different (e.g., the word *red* and blue ink). Difficulty level of the SCWT was varied according to response time allowed for each trial: the easy condition allowed 2500 m/sec and the hard condition 1500 m/sec.

Procedure

Participants were tested individually. The laboratory was sound attenuated and temperature controlled (24°C). Half of the participants were tested in the morning (8:30 AM to noon) and half in the afternoon (2:00 PM–5:30 PM) to control for any circadian differences in physiologic and performance measures. Counterbalancing was across group and gender. Participants were requested to refrain from eating and caffeine or nicotine ingestion for 1 hour before testing. Compliance was checked when each subject presented at the laboratory. Each participant in the first data collection period was tested twice, in autumn and then again

in winter. The second data collection period was conducted the following year, with each participant tested first in winter and then again in spring. Each testing session was conducted within a 14-day period as close as possible after the autumn equinox, winter solstice, or spring equinox according to the testing session. All participants were tested at the same time of day for their second session.

The experimenter recorded psychophysiological responses from an adjoining room. Height and weight measurements were taken. Heart rate and SC electrodes were attached, and the respiration band fitted around participant's upper chest. The participant then relaxed in an armchair and electrode leads were connected into MacLab. Blood pressure and sublingual temperature were taken and the participant completed BDIadd and WMI. Breathing was paced to 10 breaths/min throughout the session. A visual check of the respiratory record ensured compliance with respiratory pacing.

Baseline physiologic measurements were recorded for a period of approximately 2 min, and then tasks (SCWT and Digit Span) were given in counterbalanced order. Standard instructions from the WAIS-R manual were given for the Digit Span subtest. At completion of stressor tasks, the respiration band and finger electrodes were removed and fitness determined. The workload on the bicycle ergometer was set according to a participant's usual level of physical activity. Heart rate was monitored during the 6-min submaximal fitness test.

Data Scoring

Body mass index was calculated using the formula mass (kg)/height² (m). Respiratory sinus arrhythmia was calculated using the peak-trough method (Porjes and Byrne 1992): the difference between maximum and minimum HRs associated with each respiratory cycle and averaged over five consecutive breaths was used as an index of mean RSA. Mean HR and SCL were taken from the same five-breath period. A measure of O₂ uptake was calculated from the last 30 sec HR in the submaximal fitness test using a nomogram according to Åstrand (1960).

Results

Analyses

The winter data from both collection periods were combined, and autumn and spring data were also combined, to provide a comparison between a winter and nonwinter assessment period. For depression measures (BDIadd and WMI), 2 × 2 (Group × Season) mixed analyses of variance (ANOVAs) were performed. For RSA, HR, and SCL, preliminary analyses showed no significant differences between the four cognitive stressors (easy and hard conditions of SCWT, Forward and Backward Digit Span). The physiologic responses were averaged for the four stressors to give a baseline measure and the average physiologic physiological response for the stressors, and analyzed by 2 × 2 × 2 (Group × Season × Task Level) ANOVAs. For constitutional measures (systolic and dia-

Table 1. Means and SDs for Self-Rated Depression (Beck Depression Inventory with Addendum) and Weekly Mood Inventory Subscales for Subsyndromal Seasonal Affective Disorder (SAD) and Control Groups in Winter and Nonwinter

	Winter				Nonwinter			
	Control		SAD		Control		SAD	
	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
Beck Depression Inventory with Addendum	4.46	(4.29)	14.03	(9.39)	5.00	(5.16)	7.90	(5.81)
Weekly Mood Inventory								
Depressed Mood and Ideation (6 items)	6.64	(5.36)	14.14	(8.28)	8.50	(7.29)	11.21	(7.04)
Typical Vegetative Symptoms (5 items)	6.64	(6.14)	11.45	(7.90)	6.79	(6.62)	12.00	(5.79)
Atypical Vegetative Symptoms (5 items)	7.93	(4.34)	15.79	(6.13)	8.14	(4.19)	13.10	(5.65)
Hypomania Mood and Ideation (10 items)	36.61	(7.55)	27.41	(13.00)	37.32	(8.75)	34.31	(9.19)

Weekly Mood Inventory ratings were made on a seven-point scale and totaled for each factor. $n = 28$ control and 29 subsyndromal SAD participants.

systolic blood pressure, sublingual temperature, fitness, and BMI) 2×2 (Group \times Season) ANOVAs were performed. The α level was set at .05 for all analyses. Where analyses revealed a significant interaction, post hoc two-tailed t tests were performed to identify the source of interaction with Bonferroni adjustment (.05/ n) used (Maxwell and Delaney 1990). Between-group differences and season differences were tested with independent sample and paired sample t tests, respectively. Order effects for season were further removed statistically to ensure there were no systematic effects from winter or nonwinter testing by including a factor for order in the statistical model (Maxwell and Delaney 1990). Significant effects for order were detected in the AVS scale of the WMI, HR, RSA, and systolic and diastolic blood pressure. Reported F values indicate values after statistical adjustment of any order effects were made. All other dependent variables were nonsignificant for order.

Psychologic Analyses

Self-rated depression scores for BDIadd and WMI are shown in Table 1. For BDIadd, ANOVA revealed a main effect for Group [$F(1,55) = 19.16, p < .001$]. Seasonal affective disorder participants rated depression significantly higher than control subjects irrespective of season. There was a main effect for Season [$F(1,53) = 8.89, p = .004$], indicating that significantly greater depression ratings are recorded for winter than for nonwinter. A significant Group \times Season interaction [$F(1,55) = 12.50, p = .001$] showed SAD and control groups to vary differentially across season. Post hoc t test analyses show that SAD subjects rate depression significantly greater than control subjects in winter [$t(55) = 4.98, p < .001$] and are significantly more depressed in winter than in nonwinter [$t(28) = 3.56, p = .001$], as shown in Table 1. Control subjects do not differ significantly across seasons. These differences remain significant after applying the Bonferroni adjustment ($\alpha = .05/4 = .0125$).

With ANOVA, significant differences were shown be-

tween SAD and control groups for DMI [$F(1,55) = 13.41, p = .001$], TVS [$F(1,55) = 11.51, p = .001$], AVS [$F(1,55) = 32.23, p < .001$], and HMI [$F(1,55) = 8.93, p = .004$]. From Table 1, the SAD group rated DMI, TVS, and AVS significantly higher than control subjects, whereas HMI was rated significantly lower by the SAD group. For AVS and HMI, ANOVA also shows main effects for Season [$F(1,53) = 4.08, p = .049$, and $F(1,53) = 5.26, p = .023$, respectively]. Atypical Vegetative Symptoms are rated significantly higher and HMI significantly lower in winter. For AVS there was a significant two-way interaction between Group and Season [$F(1,53) = 5.40, p = .024$], indicating the groups vary differentially across seasons. Post hoc t test analyses show significant group differences. Seasonal affective disorder subjects rate symptoms significantly higher than control subjects for both winter [$t(55) = 5.57, p < .01$] and nonwinter [$t(55) = 3.76, p < .01$] (Table 1). These seasonal differences remain significant after the Bonferroni adjustment is applied ($\alpha = .05/4 = .0125$). There is also a strong seasonal tendency for SAD subjects to rate symptoms higher in winter than in nonwinter [$t(28) = 1.939, p = .06$].

Psychophysiologic Analyses

RSA. There was a significant decrease in RSA between base and stressor tasks [$F(1,161) = 14.15, p < .001$], and RSA is significantly lower for stressor tasks (mean = 13.76, SD = 5.23) than baseline (mean = 15.67, SD = 7.15). Analysis of variance also showed a significant two-way interaction between Group and Season [$F(1,161) = 6.37, p = .01$]. Seasonal affective disorder and control groups varied differentially in RSA. This interaction is shown in Figure 1.

Post hoc t test analyses were performed between groups and seasons. Figure 1 shows SAD subjects to have higher RSA for winter (mean = 15.95, SD = 4.44) than for nonwinter (mean = 13.52, SD = 5.36) [$t(28) = 2.34, p = .026$]. This difference for control subjects is nonsignifi-

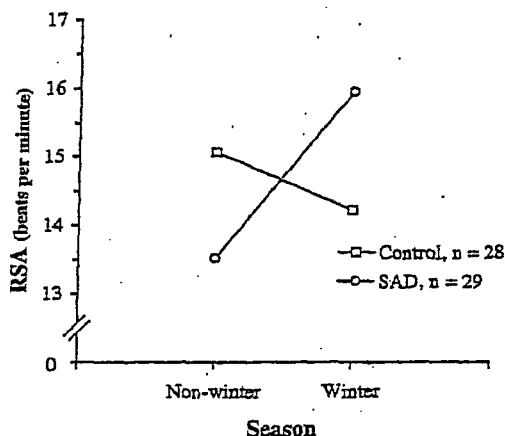


Figure 1. Group mean respiratory sinus arrhythmia during non-winter and winter for subsyndromal seasonal affective disorder (SAD) and control participants. RSA, respiratory sinus arrhythmia.

cant. The differences between groups in winter and in nonwinter were nonsignificant. When the conservative Bonferroni adjustment is applied ($\alpha = .05/4 = .0125$), the difference in RSA for SAD subjects from winter to nonwinter is no longer significant.

HR. The main effect for Task Level [$F(1,161) = 35.35, p < .001$] indicates a highly significant increase in HR from baseline (mean = 73.90, SD = 10.79) to stressor tasks (mean = 79.13, SD = 9.99) (Figure 2).

The main effect for Season [$F(1,158) = 3.89, p = .05$] indicates that HR is significantly lower for winter

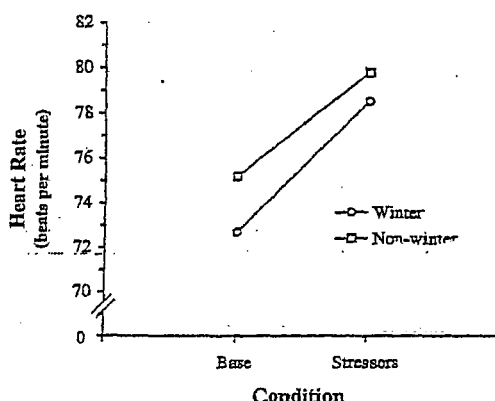


Figure 2. Group mean heart rate (beats/min) during winter and nonwinter for subsyndromal seasonal affective disorder and control participants combined.

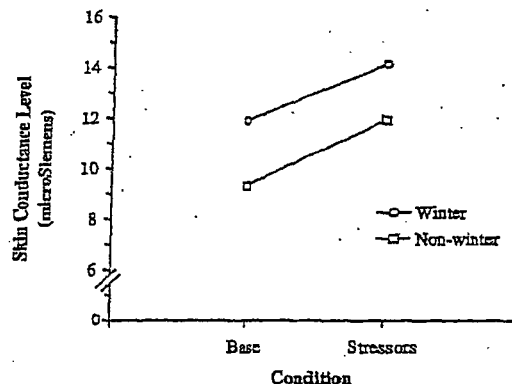


Figure 3. Group mean skin conductance level during winter and nonwinter for subsyndromal seasonal affective disorder and control participants combined.

(mean = 75.59, SD = 10.17) than for nonwinter (mean = 77.47, SD = 10.60).

SCL. The main effect for Task Level (Figure 3) [$F(1,158) = 22.64, p < .001$] indicates that SCL increases significantly from base (mean = 10.62, SD = 6.26) to stressors (mean = 13.04, SD = 6.58) (Figure 3).

The main effect for Season [$F(1,158) = 21.82, p = .001$] indicates SCL is higher for winter (mean = 13.03, SD = 6.94) than for nonwinter (mean = 10.63, SD = 5.89).

Constitutional Variable Analyses

For diastolic blood pressure, ANOVA showed a significant main effect for Season [$F(1,53) = 5.45, p = .023$]. Diastolic blood pressure is significantly higher in winter (mean = 74.96, SD = 8.15) than in nonwinter (mean = 73.23, SD = 6.97). Analyses of systolic blood pressure, sublingual temperature, BMI, and submaximal fitness showed no significant main effects or interactions.

Discussion

Seasonal affective disorder participants reported significantly higher depression and seasonal symptom ratings than control subjects for winter than for nonwinter. Significant interactions for BDI and the WMI factor for SAD symptoms, AVS, show higher ratings for depression and associated symptoms for SADs in winter. Atypical Vegetative Symptoms showed a nonsignificant seasonal tendency for SAD subjects to rate symptoms higher in winter than in nonwinter. Typical Vegetative Symptoms, the scale for nonseasonal symptoms of depression, did not differ between seasons.

The significant interaction between group and season for RSA, together with the significant main effect for season for HR, offer support for the hypothesis that SAD subjects have higher RSA, or greater vagal tone, in winter. The significant interaction for RSA is due to the significantly higher winter scores for SAD subjects. However, after Bonferroni correction this effect became nonsignificant and should be replicated as the type 2 error rate is greatly increased by Bonferroni. Main effects for Task Level show RSA to decrease, whereas HR and SCL increase significantly from baseline to stressors for both groups as expected. Combined groups show a decrease in RSA and increases in HR and SCL in winter.

Seasonality is evident for control subjects showing a lower HR in winter. Respiratory sinus arrhythmia does not vary between seasons for control subjects. These differences in RSA and HR cannot be accounted for by fitness level. Submaximal fitness tests found no significant differences between SAD and control groups or between seasons. Seasonality is also evident in SCL and blood pressure. A winter peak has been shown previously in SCL (Neumann 1968; Venables and Christie 1973) and is supported by the present study. Blood pressure has also been shown to peak in winter for normal subjects (Lacoste and Witz-Justice 1989). The present study shows diastolic blood pressure to be higher in winter. No differences were shown for systolic blood pressure.

Several implications can be made from the study. First, it indicates that SAD subjects have increased RSA in winter. This increase in vagal tone may reflect the sleep symptom in SAD, thus showing a similarity with the hibernation process. Seasonal affective disorder has been compared to animal models of hibernation (Rosenthal et al 1984), with similarities including hypersomnia, overeating, change in food preference, and weight gain. Although this does not imply similar underlying mechanisms, the comparison may assist in identifying similarities and differences in SAD and hibernating mechanisms. Burlington and Milsom (1989) have identified the parasympathetic nervous system as having an involvement in the hibernation process. Further, a suppression of sympathetic activity during hibernation has been reported in Syrian hamsters (Wade 1989) and is possibly implicated for SAD participants in the present study. On the other hand, nonseasonal major depressive disorder has been associated with decreased vagal tone (Balogh et al 1993), which may indicate different mechanisms between the two disorders.

Second, the reduction in HR during winter suggests that, for SAD participants, this is consistent with increased vagal tone but may also be due to reduced sympathetic nervous system arousal. Previous research indicates that disruption of noradrenergic pathways (Shafi and Shafi 1990) may implicate noradrenaline in the pathogenesis of

SAD. The present finding supports several other research findings. These are 1) low levels of noradrenaline in SAD subjects relative to normal subjects during winter (Hill 1992; Shafi and Shafi 1990; Skwerer et al 1989), 2) significant positive correlations reported between increases in noradrenaline after light therapy and decreases in seasonal depression (Skwerer et al 1989), and 3) no improvement (Hill 1992) or a worsening (Schlager 1994) in winter depression found after treatment with β -blockers.

A third implication from the study relates to the seasonality shown in variables for all participants regardless of group. Lower HR was shown for winter, whereas SCL and diastolic blood pressure were shown to peak for winter, suggesting caution is required when comparing these variables across seasons. Research across four seasons is necessary to provide data showing the extent of seasonal changes in physiologic responses for control subjects, thus extending our knowledge about circannual changes.

In conclusion, SADs have increased vagal tone in winter. Comparison with the hibernation process in animals may help explain SAD and show that SAD is distinct from nonseasonal depression. Further research across a year-long period is being conducted with additional measures to confirm and extend the differential physiologic response changes across groups and find out which would support the view of the syndrome as being a disorder distinct from nonseasonal depression.

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